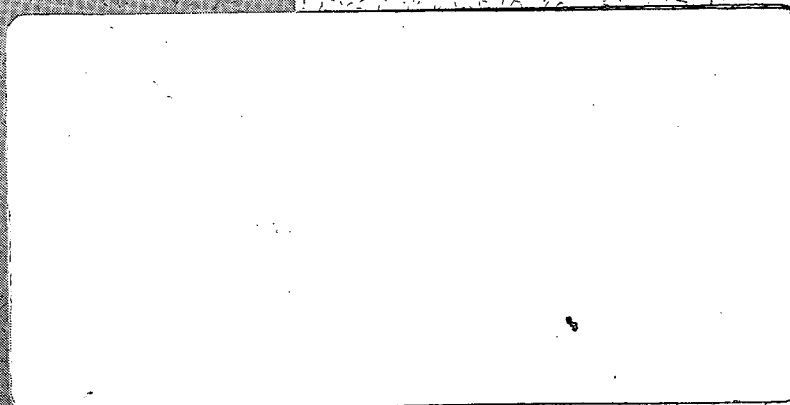# REAC

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OFFICE OF EMERGENCY AND REMEDIAL RESPONSE

97P-1159

Final Report (Vol III Appendix D-H)
Cornell-Dubilier Site
South Planfield, NJ
April 1998

APPENDIX D

Amphipod Toxicity Evaluation Final Report
Cornell-Dubilier Site
South Plainfield, NJ
April 1998

FINAL REPORT

TOXICOLOGICAL EVALUATION OF SEDIMENT
COLLECTED AT THE CORNELL DUBILIER
ELECTRONICS SITE, SOUTH PLAINFIELD, NJ

November 22, 1997

REPORT #: 7286

PREPARED FOR:

WESTON - REAC
GSA RARITAN DEPOT
BUILDING 209
EDISON, NJ 08837

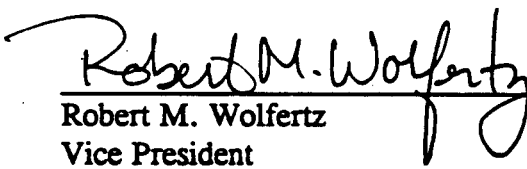|	
Report:	Toxicological Evaluation of Sediment Collected from Cornell Dubilier Electronics Site, South Plainfield, New Jersey
Sponsor:	Weston - REAC GSA Raritan Depot Building 209 Edison, NJ 08837
Testing Facility:	Aqua Survey, Inc. 499 Point Breeze Road Flemington, NJ 08822 USA
Study Number:	97-286
Report:	7286
Study Initiation Date:	June 21, 1997
Date Reported:	November 22, 1997
Personnel:	A. Novotny S. Hornberger J. Rios T. Dolce C. Doyle E. Landon M. Papalski Y. Terrell

Robert M. Wolfertz   11/21/97

Robert M. Wolfertz   Date
Vice President

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I.   Introduction

The objective of this test was to determine the toxic effects of sediment collected from the Cornell Dubilier Electronics Site in South Plainfield, New Jersey, to the benthic invertebrate, *Hyalella azteca*. The measure of toxicity is a statistically significant reduction in survival and/or growth of exposed organisms as compared to a reference following exposure for 14 days. Survival in all samples following the 14-day exposure ranged from 76.7% to 94.2%. Survival in sample A3-1 was statistically significant when compared to that of the reference, sample A9-1, indicating acute toxicity. There was no statistically significant reduction in growth of the amphipods ($p \leq 0.05$) when compared to the control or reference, indicating no measurable chronic toxicity to the amphipod *H. azteca*.

Table 1: Survival and Growth Results from 14-day Solid Phase Toxicity Test with *H. azteca*.

Sample ID	ASI ID	Location	% Survival	Mean Length in mm.
Pretest	n/a	n/a	n/a	1.4
Control	71174	n/a	83.3	2.7
J10265 (ref.)	71164	A9-1	91.7	2.4
J10264	71165	A3-1	76.7*	2.7
J10262	71166	A6-2	88.3	2.7
J10497	71167	A5-2	82.5	2.5
J10494	71168	A4-1	90.8	2.4
J10493	71169	A2-2	94.2	2.8
J10486	71170	A1-1	90.8	2.8

* = Statistically significant, compared to Reference, $p \leq 0.05$.

II.   Materials and Methods

All samples were collected by Weston personnel from the Cornell Dubilier Electronics Site in South Plainfield, New Jersey, and were received in acceptable condition at Aqua Survey on June 21, 1997. A control sediment was prepared at Aqua Survey, sample A9-1 was used as the reference sediment, and the test sediments were samples A3-1, A6-2, A5-2, A4-1, A2-2, and A1-1.

The methods employed in this test followed guidelines outlined in the following references:

American Public Health Association. 1985. Standard Methods for the Examination of Water and Wastewater, 16th ed., 379 pp.

Nebeker et al. 1984. Biological Methods for Determining Toxicity of Contaminated Freshwater Sediments to Invertebrates, Envir. Tox. Chem,. Vol 3: 617-630.

U.S. EPA, 1979, Handbook for Analytical Quality Control in Water and Wastewater Laboratories, EPA/600/4-79/019.

III.   Test Conditions

Species
The test species for this test was *Hyalella azteca*, which is a representative benthic invertebrate.

Size/Age/Physical Condition
Animals used in this test were collected from a mass culture. The exact age could not be determined. However, based on the length of the organisms (1.0-2.1 mm), their age should be approximately 0 - 7 days old. The organisms appeared to be in good condition.

Source/Acclimation
All of the animals used were obtained from ASI's in-house culture. Overlay water used was the same as the culture water, therefore no acclimatization was necessary.

Source of Overlay Water
Overlay water was ASI culture water (well water).

Test Temperature
23 ± 1°C

Test Vessels
The test vessels were new 1-L nalgene beakers covered with a petri plate and beaker fill attachment. Aeration was provided through a filtered, forced air system and plastic-tipped airlines.

Photoperiod
The test was conducted on a 16-hour light/8-hour dark photoperiod with two, 30-minute phase-in/phase-out periods.

Test Levels
Six replicates of undiluted test sediment were tested and compared to six replicates of control sediment.

Reference
Reference sediment was tested concurrently with the test samples.

Performance Controls
Performance controls are designed to assess the quality of the test organisms and the capabilities of the testing laboratory to successfully perform the toxicity tests. The sediment for performance controls was a mixture of sediment collected from ASI's culture ponds and washed silica sand. ASI culture water (well water) was used as the overlay water during testing. Controls were run with the same number of organisms as the test samples.

IV. Test Procedures

Sediment Sample Preparation
All samples were sieved by pressing the mixed material through a 2-mm sieve. Materials remaining on the sieve were discarded. Samples were homogenized with a plastic spoon by hand until uniform in texture and consistency.

Approximately 200 mL of test sediment was placed into each of six replicate test vessels. The vessels were then filled with dilution water using a head chamber fitted with a 1/4-inch ID drain tube. The tube was bent slightly at the bottom to divert the water away from the sediment. The system was allowed to settle under gentle aeration overnight before organisms were added.

Beginning the Test
Test chambers were monitored for temperature, pH, and dissolved oxygen using a YSI multi-parameter probe. Total ammonia was measured in every chamber using an Orion 290A ion-specific meter fitted with an Orion 95-12 ammonia probe. Conductivity was determined with a YSI Model 33 conductivity meter on a composite sample of each of the eight replicates. Alkalinity and hardness were also determined in the composite sample using standard titrametric methods.

Animals were chosen at random from the culture and counted into 30-mL transport cups. Ten organisms were added to each cup. Once all the cups had ten organisms, organism counts were verified by a second person and the cups were then randomly assigned to chambers. The test began when organisms were introduced to all chambers.

Daily Maintenance
Dissolved oxygen, pH, and temperature were determined daily in every test vessel using a YSI 600 multi-parameter probe and a YSI 610-DM data logger. All test chambers were examined daily and observed behaviors noted. Total ammonia readings were taken each day from the overlay water in one representative sample from each treatment. Animals were fed one mL of standard YCT solution every other day.

Test Duration
The sediment toxicity test was 14 days in duration.

Ending the Test

At the conclusion of the test, dissolved oxygen, temperature, pH and ammonia were measured in each beaker. A composite of the overlay water from each treatment was analyzed for conductivity, alkalinity, and hardness.

Animals were removed by sieving the sediment through a 500-µm mesh screen using a minimal amount of fresh overlay water. The amphipods retained on the screen were removed with a pipet. The number of survivors was noted, and survivors were euthanized in a solution of 70% methanol. Once dead, the amphipods were measured to the nearest 0.1 mm using a dissecting microscope and micrometer.

Statistical Analyses

At the conclusion of the test, the data were entered into the statistical software, Toxstat™ as appropriate. Survival data were transformed using arcsine squareroot and tested for normality and homogeneity. Based on the results, parametric or non-parametric analysis of variance and comparison of means were used to determine which samples were statistically significant from the reference. Those treatments exhibiting either no or extremely low survival were excluded from the analysis. Growth data were entered into the database untransformed, and tested for normality and homogeneity. The data were then analyzed using analysis of variance and Dunnett's multiple comparison test to determine which organisms were statistically significant from the control and the reference.

Reference Toxicant

A standard reference toxicant (SRT) test with cadmium chloride was performed. The toxicant was dissolved in dilution water to make a stock solution which was diluted to achieve the desired concentration. A substrate of nylon mesh screen was provided.

V. Results

Survival

Average survival ranged from 76.7% to 94.2% all treatments. Performance control survival was 83.3%, which exceeds the USEPA and ASTM minimum criteria of 80%. Sample A3-1 was the only sample shown to be significantly significant from the reference, and was acutely toxic to the amphipod, *Hyalella azteca* after a fourteen day exposure.

Growth

The organisms used in testing ranged from 1.0 to 2.1 mm in length at the start of the test. Surviving organisms averaged from 1.4 to 5.3 mm in length in the various treatments by the end of the test. Data from replicate A3-1 were not included in the calculation of statistics for length, as the survival in that replicate was statistically significant from the reference. There was no statistically significant difference between the organisms exposed to test sediments and those exposed to either the control or reference sediment, therefore we report no measureable chronic toxicity to the amphipod, *Hyalella azteca* in a fourteen day exposure.

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Water Quality
The target test temperature was 22.0 to 24.0°C. The test solution temperature ranged from 22.0 to 24.0°C. The target dissolved oxygen was greater than 4.0 mg/L. Dissolved oxygen was maintained at or above 4.2 mg/L in all chambers. Aeration was commenced prior to introduction of organisms on day 0 due to an expected decline in dissolved oxygen levels. The pH ranged from 7.0 to 9.1. Total ammonia in the overlay water ranged from undetected to 3.26 mg/L (MDL 0.08 mg/L). Although the total ammonia in some of the test vessels changed by greater than 50 percent, the maximum difference recommended by the U.S.EPA test guidelines, this change is believed not to have caused the observed toxicity. Alkalinity ranged from 180 to 260 mg/L $CaCO_3$. Specific conductance ranged from 520 - 620 umhos/cm. Hardness ranged from 112 to 164 mg/L $CaCO_3$. Based on previous tests with this organism, none of these water quality indicators are sufficient to explain the observed mortality.

Complete water quality data are presented in Tables 3 - 7 at the end of this section.

Standard Reference Toxicant
The 96-hour LC_{50} for the reference toxicant was determined to be 8.33 μg Cd/L. This is within the expected range of 0 - 42.7 μg/L for this organism at ASI. A summary of the live count data for this test is presented below in Table 2, and the water quality parameters as well as a control chart are included in the appendix.

Table 2: Results of 96-hour Standard Reference Toxicant Test with $CdCl_2$
Tests were initiated with 10 *Hyalella azteca* per concentration.

Concentration (μg Cd/L)	Final Live Counts
Control	8
4.0	8
8.0	6
16.0	2
32.0	0
64.0	0

LC_{50} = 8.33 (5.40 - 11.9)μg Cd/L

VI. Source of Documentation

All original data will be maintained at:
      Aqua Survey, Inc.
      499 Point Breeze Road
      Flemington, NJ 08822

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AQUA SURVEY, INC.
14-DAY SOLID PHASE READINGS

Table 3

CLIENT:	Rov F. Weston	TEST DATE:	6/25/97	PARAMETER: Live Count
JOB #:	97-286	INITIAL COUNT:	20	ORGANISM: *H. azteca*

Position #	ID#	Sample	Final Count	Percent Survival
43	0.1	Control	16	
30	0.2		17	
27	0.3		17	
41	0.4		16	
26	0.5		16	
10	0.6		18	83.3%
44	1.1	J10265	19	
25	1.2	A9-1	20	
1	1.3		19	
9	1.4		16	
23	1.5		18	
24	1.6		18	91.7%
48	2.1	J10264	17	
33	2.2	A3-1	14	
19	2.3		14	
2	2.4		17	
22	2.5		17	
18	2.6		13	76.7% *
46	3.1	J10262	20	
15	3.2	A6-2	17	
47	3.3		17	
35	3.4		17	
16	3.5		17	
38	3.6		18	88.3%
8	4.1	J10497	17	
39	4.2	A5-2	15	
21	4.3		11	
40	4.4		18	
45	4.5		20	
11	4.6		18	82.5%
32	5.1	J10494	20	
20	5.2	A4-1	19	
42	5.3		17	
14	5.4		20	
3	5.5		17	
31	5.6		16	90.8%
7	6.1	J10493	19	
5	6.2	A2-2	17	
29	6.3		20	
28	6.4		19	
17	6.5		19	
12	6.6		19	94.2%
6	7.1	J10486	17	
37	7.2	A1-1	18	
4	7.3		18	
34	7.4		17	
36	7.5		19	
13	7.6		20	90.8%

* Statistically significant, compared to the Reference, $p \leq 0.05$

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AQUA SURVEY, INC.
SOLID PHASE READINGS

Table 4
CLIENT: Roy F. Weston   TEST START DATE: 6/25/97   PARAMETER: Temp. (degrees C)
JOB #: 97-286   ORGANISM: H. azteca

Positio	ID#	Sample	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Low	High
43	0.1	Control	23.9	22.7	22.6	22.6	22.6	22.7	22.6	22.5	22.6	22.8	22.4	22.5	22.8	22.5	22.6		
30	0.2		24.0	22.7	22.7	22.6	22.8	22.9	22.9	22.8	22.9	22.7	22.4	22.2	23.0	22.5	22.4		
27	0.3		24.0	22.5	22.9	22.8	23.0	23.0	23.1	22.7	22.7	22.6	22.4	22.5	23.1	22.8	22.6		
41	0.4		24.0	22.7	22.8	22.7	22.8	22.8	22.7	22.7	22.8	22.7	22.5	22.4	22.9	22.5	22.6		
26	0.5		24.0	22.6	22.8	22.7	22.9	22.9	22.9	22.6	22.7	22.5	22.4	22.4	22.9	22.6	22.6		
10	0.6		23.9	22.5	22.7	22.6	22.8	22.8	22.7	22.7	22.7	22.6	22.5	22.4	22.8	22.3	22.5	22.2	24.0
44	1.1	J10265	23.7	22.7	22.7	22.7	22.8	22.8	22.8	22.7	22.7	22.8	22.3	22.5	22.9	22.4	22.6		
25	1.2	A9-1	24.0	22.7	22.7	22.7	22.8	22.8	22.9	22.7	22.5	22.7	22.4	22.3	22.8	22.4	22.6		
1	1.3		23.9	22.6	22.6	22.5	22.5	22.6	22.5	22.3	22.5	22.5	22.4	22.3	22.4	22.0	22.0		
9	1.4		24.0	22.7	22.8	22.5	22.8	22.9	22.8	22.7	22.8	22.7	22.5	22.4	23.2	22.5	22.5		
23	1.5		24.0	22.8	22.8	22.8	22.9	22.9	22.9	22.6	22.7	22.6	22.4	22.4	22.9	22.5	22.6		
24	1.6		23.9	22.6	22.8	22.8	22.9	22.7	22.9	22.7	22.6	22.7	22.4	22.2	22.7	22.5	22.6	22.0	24.0
48	2.1	J10264	23.9	22.7	22.9	22.7	22.9	23.0	22.9	22.7	22.7	22.8	22.6	22.6	22.9	22.6	22.6		
33	2.2	A3-1	24.0	22.7	22.8	22.8	22.8	22.9	22.9	22.7	22.7	22.7	22.6	22.6	23.0	22.4	22.5		
19	2.3		24.0	22.8	22.8	22.7	22.9	22.8	22.8	22.7	22.7	22.7	22.4	22.4	23.0	22.4	22.5		
2	2.4		23.9	22.3	22.6	22.4	22.6	22.6	22.5	22.4	22.6	22.5	22.3	22.4	22.5	22.1	22.1		
22	2.5		24.0	22.7	22.7	22.7	22.9	22.8	22.8	22.5	22.7	22.6	22.3	22.2	22.8	22.5	22.5		
18	2.6		24.0	22.7	22.8	22.7	22.9	22.7	22.7	22.7	22.7	22.6	22.2	22.4	22.7	22.4	22.5	22.1	24.0
46	3.1	J10262	23.9	22.8	22.8	22.7	22.8	22.9	22.8	22.7	22.7	22.8	22.5	22.6	23.0	22.5	22.6		
15	3.2	A6-2	24.0	22.7	22.8	22.6	22.9	22.8	22.7	22.6	22.7	22.6	22.3	22.4	22.9	22.4	22.5		
47	3.3		24.0	22.8	22.7	22.6	22.7	22.9	22.8	22.6	22.8	22.8	22.5	22.4	22.7	22.5	22.6		
35	3.4		23.9	22.7	22.8	22.8	22.8	22.8	22.8	22.6	22.6	22.7	22.5	22.5	22.9	22.5	22.5		
16	3.5		24.0	22.7	22.7	22.7	22.8	22.8	22.7	22.6	22.7	22.7	22.3	22.5	22.7	22.4	22.5		
38	3.6		24.0	22.8	22.8	22.8	22.8	22.9	22.8	22.7	22.7	22.7	22.4	22.4	23.0	22.4	22.5	22.3	24.0
8	4.1	J10497	24.0	22.8	22.7	22.5	22.8	22.8	22.6	22.6	22.7	22.7	22.4	22.4	23.0	22.4	22.4		
39	4.2	A5-2	23.9	22.7	22.8	22.7	22.8	22.9	22.7	22.7	22.7	22.8	22.5	22.3	22.9	22.5	22.5		
21	4.3		24.0	22.7	22.8	22.6	22.9	22.8	22.8	22.7	22.7	22.7	22.5	22.3	22.8	22.4	22.5		
40	4.4		24.0	22.6	22.8	22.7	22.7	22.9	22.8	22.7	22.7	22.8	22.6	22.5	22.9	22.5	22.6		
45	4.5		23.7	22.7	22.7	22.7	22.8	22.9	22.7	22.7	22.8	22.7	22.4	22.5	22.9	22.5	22.6		
11	4.6		24.0	22.6	22.7	22.6	22.7	22.8	22.7	22.7	22.7	22.6	22.5	22.5	22.8	22.4	22.5	22.3	24.0
32	5.1	J10494	24.0	22.7	22.7	22.7	22.8	22.9	22.9	22.7	22.8	22.8	22.6	22.6	23.0	22.5	22.5		
20	5.2	A4-1	24.0	22.9	22.8	22.6	22.8	22.8	22.8	22.7	22.7	22.7	22.5	22.4	22.8	22.4	22.5		
42	5.3		23.9	22.7	22.8	22.8	22.8	22.8	22.8	22.7	22.7	22.7	22.5	22.4	22.9	22.5	22.6		
14	5.4		24.0	22.6	22.7	22.6	22.8	22.8	22.6	22.5	22.7	22.6	22.4	22.3	22.9	22.3	22.5		
3	5.5		23.9	22.2	22.6	22.3	22.6	22.7	22.7	22.6	22.7	22.6	22.2	22.2	23.0	22.2	22.2		
31	5.6		24.0	22.7	22.8	22.8	22.9	22.9	22.9	22.7	22.8	22.7	22.5	22.5	22.9	22.5	22.5	22.2	24.0
7	6.1	J10493	24.0	22.7	22.7	22.5	22.8	22.8	22.8	22.6	22.7	22.6	22.3	22.3	23.3	22.4	22.4		
5	6.2	A2-2	24.0	22.7	22.7	22.4	22.7	22.8	22.7	22.6	22.7	22.7	22.2	22.4	23.1	22.1	22.3		
29	6.3		24.0	22.8	22.8	22.8	22.9	22.8	22.8	22.7	22.8	22.5	22.4	22.4	22.7	22.5	22.3		
28	6.4		24.0	22.7	22.8	22.8	22.9	23.0	22.9	22.7	22.8	22.7	22.3	22.5	23.2	22.7	22.6		
17	6.5		24.0	22.7	22.8	22.7	22.8	22.8	22.7	22.6	22.6	22.7	22.4	22.4	22.9	22.4	22.5		
12	6.6		24.0	22.8	22.7	22.6	22.7	22.7	22.7	22.6	22.6	22.7	22.2	22.5	23.0	22.3	22.5	22.1	24.0
6	7.1	J10486	24.0	22.6	22.7	22.5	22.8	22.8	22.6	22.6	22.7	22.5	22.1	22.1	22.7	22.2	22.4		
37	7.2	A1-1	24.0	22.7	22.7	22.7	22.8	22.9	22.8	22.6	22.7	22.7	22.5	22.6	23.0	22.5	22.5		
4	7.3		24.0	22.7	22.6	22.5	22.6	22.8	22.6	22.5	22.6	22.7	22.1	22.2	22.7	22.3	22.3		
34	7.4		24.0	22.6	22.9	22.8	23.0	22.9	22.9	22.7	22.7	22.7	22.6	22.6	23.0	22.4	22.5		
36	7.5		23.9	22.7	22.8	22.7	22.9	22.9	22.8	22.7	22.7	22.8	22.6	22.6	23.0	22.5	22.5		
13	7.6		24.0	22.5	22.7	22.7	22.8	22.8	22.7	22.6	22.6	22.7	22.4	22.5	23.1	22.4	22.5	22.1	24.0

Ranges   22.0   24.0

Table 5

CLIENT: Roy F. Weston   TEST START DATE: 6/25/97   PARAMETER: D.O. (mg/L)
JOB #: 97-286   ORGANISM: H. azteca

Position #	ID#	Sample	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Low	High
43	0.1	Control	6.4	7.7	7.7	7.7	7.8	7.6	7.6	8.2	7.9	7.3	7.6	7.9	7.7	8.1	7.5		
30	0.2		6.6	7.7	7.5	7.5	7.4	7.4	7.7	7.8	7.3	7.1	7.5	7.8	7.1	8.1	7.4		
27	0.3		6.4	7.9	7.2	7.5	7.5	7.4	7.5	7.6	7.7	7.3	7.7	8.0	7.8	8.0	7.4		
41	0.4		6.2	7.7	7.6	7.7	7.7	7.6	7.5	8.1	7.8	7.3	7.6	7.9	7.9	8.1	7.4		
26	0.5		6.5	7.7	7.5	7.6	7.5	7.4	7.6	7.6	7.8	7.3	7.7	8.1	7.9	8.1	7.4		
10	0.6		6.9	7.7	7.6	7.4	7.6	7.4	7.8	7.4	7.6	7.3	7.6	7.8	8.0	8.2	7.4	6.2	8.2
44	1.1	J10265	6.9	7.7	7.5	7.6	7.8	7.6	7.7	8.1	7.9	7.3	7.6	7.9	6.9	8.1	7.5		
25	1.2	A9-1	6.1	7.7	7.7	7.5	7.6	7.4	7.7	7.8	7.7	7.2	7.7	8.1	7.8	8.2	7.4		
1	1.3		6.0	7.8	7.8	7.8	8.1	7.3	7.5	8.5	8.2	7.1	7.5	8.0	6.6	8.3	7.7		
9	1.4		6.0	7.7	7.6	7.4	7.7	7.4	7.8	7.7	7.7	7.2	7.5	7.8	7.9	8.3	7.4		
23	1.5		5.8	7.6	7.6	7.5	7.5	7.3	7.5	7.7	7.7	7.2	7.7	8.1	7.2	8.2	7.4		
24	1.6		6.1	7.7	7.7	7.5	7.6	7.5	7.7	7.7	7.7	7.2	7.7	8.1	7.8	8.2	7.4	5.8	8.5
48	2.1	J10264	6.0	7.7	7.5	7.6	7.6	7.4	7.8	8.1	7.8	7.4	7.6	7.9	7.9	8.3	7.5		
33	2.2	A3-1	5.6	7.7	7.7	7.6	7.7	7.4	7.7	8.1	7.9	7.2	7.4	7.7	7.7	8.1	7.4		
19	2.3		4.2	7.7	6.6	7.5	7.5	7.3	7.5	7.5	7.5	7.3	7.8	8.1	8.0	8.3	7.4		
2	2.4		4.5	7.5	7.7	7.7	8.0	7.3	6.5	8.2	8.0	7.2	7.5	7.9	7.4	8.1	7.6		
22	2.5		5.0	7.6	7.6	7.6	7.6	7.4	7.6	7.7	7.7	7.2	7.7	8.1	8.0	8.2	7.4		
18	2.6		6.9	7.7	6.4	7.5	7.6	7.4	7.7	7.5	7.6	7.3	7.8	8.1	8.0	8.3	7.4	4.2	8.3
46	3.1	J10262	6.6	7.7	7.6	7.7	7.7	7.6	7.9	8.2	7.9	7.4	7.6	7.9	7.8	8.1	7.5		
15	3.2	A6-2	7.1	7.7	7.5	7.5	7.5	7.3	7.8	8.1	7.7	7.3	7.7	8.0	7.7	8.3	7.4		
47	3.3		6.8	7.7	7.6	7.6	7.8	7.5	7.9	8.2	7.9	7.4	7.6	7.9	7.9	8.2	7.5		
35	3.4		6.0	7.6	7.4	7.5	7.5	7.5	7.5	7.9	7.8	7.2	7.5	7.8	7.8	8.0	7.4		
16	3.5		6.6	7.7	7.6	7.6	7.6	7.3	7.8	8.1	7.8	7.3	7.7	8.1	7.9	8.3	7.4		
38	3.6		6.5	7.6	7.6	7.6	7.6	7.4	7.8	8.0	7.7	7.3	7.5	7.8	7.7	8.1	7.4	6.0	8.3
8	4.1	J10497	6.0	7.6	7.7	7.4	7.8	7.4	7.8	7.6	7.7	7.2	7.5	7.7	7.8	8.4	7.4		
39	4.2	A5-2	6.0	7.7	7.6	7.6	7.6	7.4	7.7	8.0	7.8	7.2	7.5	7.9	7.9	8.1	7.4		
21	4.3		6.5	7.6	7.7	7.6	7.6	7.3	7.5	7.7	7.6	7.2	7.7	8.1	8.0	8.4	7.4		
40	4.4		6.2	7.7	7.6	7.6	7.7	7.4	7.6	8.0	7.9	7.2	7.5	7.9	7.9	8.2	7.4		
45	4.5		6.8	7.7	7.6	7.7	7.8	7.6	7.9	8.2	7.9	7.4	7.6	7.9	7.8	8.1	7.5		
11	4.6		6.3	7.7	7.5	7.5	7.6	7.4	7.7	7.5	7.7	7.3	7.6	7.9	7.7	8.2	7.4	6.0	8.4
32	5.1	J10494	6.5	7.7	7.6	7.6	7.7	7.4	7.7	7.9	7.8	7.1	7.4	7.7	7.7	8.1	7.4		
20	5.2	A4-1	5.8	7.6	7.7	7.6	7.5	7.3	7.4	7.5	7.5	7.2	7.7	8.1	8.0	8.4	7.5		
42	5.3		6.3	7.7	7.6	7.7	7.7	7.6	7.5	8.1	7.9	7.3	7.6	7.9	7.3	8.1	7.5		
14	5.4		6.8	7.7	7.2	7.5	7.5	7.3	7.9	8.1	7.8	7.3	7.7	8.0	7.5	8.3	7.4		
3	5.5		6.0	7.6	7.6	7.7	7.9	7.3	6.6	7.7	7.9	7.2	7.5	7.9	7.3	8.0	7.5		
31	5.6		6.7	7.7	7.5	7.6	7.5	7.4	7.7	7.9	7.6	7.1	7.5	7.8	7.7	8.1	7.4	5.8	8.4
7	6.1	J10493	5.9	7.7	7.5	7.5	7.8	7.4	7.4	7.5	7.7	7.3	7.4	7.7	7.6	8.3	7.4		
5	6.2	A2-2	5.9	7.7	7.6	7.6	7.8	7.3	7.2	7.8	7.8	7.2	7.5	7.8	7.4	8.0	7.4		
29	6.3		5.9	7.7	7.6	7.5	7.4	7.5	7.8	7.7	7.4	7.2	7.6	8.4	7.8	8.0	7.4		
28	6.4		6.1	7.8	7.5	7.5	7.6	7.4	7.5	7.7	7.6	7.2	7.7	8.0	7.8	8.1	7.4		
17	6.5		7.3	7.7	7.4	7.6	7.7	7.4	7.7	8.1	7.8	7.3	7.8	8.1	7.9	8.3	7.4		
12	6.6		6.0	7.7	7.6	7.6	7.6	7.6	7.8	7.8	7.9	7.3	7.6	7.9	7.9	8.2	7.4	5.9	8.4
6	7.1	J10486	6.5	7.7	7.6	7.6	7.8	7.5	7.4	7.3	7.7	7.3	7.5	7.8	7.7	8.1	7.4		
37	7.2	A1-1	6.2	7.6	7.6	7.6	7.5	7.4	7.8	8.1	7.8	7.3	7.5	7.8	7.8	8.1	7.4		
4	7.3		6.1	7.7	7.7	7.7	7.9	7.3	7.2	7.8	7.9	7.2	7.5	7.9	7.5	8.0	7.5		
34	7.4		6.0	7.8	7.6	7.5	7.5	7.4	7.6	8.0	7.8	7.2	7.5	7.7	7.7	8.1	7.4		
36	7.5		5.8	7.6	7.2	7.5	7.5	7.5	7.5	7.9	7.8	7.2	7.5	7.8	7.8	8.1	7.4		
13	7.6		6.0	7.7	6.9	7.5	7.6	7.5	7.7	8.0	8.0	7.3	7.6	7.9	7.4	8.3	7.4	5.8	8.3
																Ranges			4.2	8.5

*Test was aerated prior to introduction of organisms on 6/25/97

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AQUA SURVEY, INC.
14-DAY SOLID PHASE READINGS

Table 6

CLIENT: Rov F. Weston TEST START DATE: 6/25/97 PARAMETER: pH (su)
JOB #: 97-286 ORGANISM: H. azteca

Position #	ID#	Sample	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Low	High
43	0.1	Control	7.3	8.2	8.1	8.3	8.1	8.2	8.3	8.3	8.3	8.3	8.7	8.6	8.6	8.3	8.7		
30	0.2		7.6	8.0	8.3	8.3	8.3	8.2	8.2	8.3	8.3	7.9	8.6	8.4	8.6	8.3	8.6		
27	0.3		7.6	8.2	8.2	8.3	8.3	8.2	8.3	8.3	8.3	8.3	8.4	8.6	8.7	8.4	8.6		
41	0.4		7.5	8.1	8.2	8.4	8.3	8.3	8.3	8.3	8.3	8.4	8.9	8.8	8.6	8.4	8.8		
26	0.5		7.5	7.9	8.1	8.3	8.2	8.1	8.2	8.2	8.2	8.3	8.4	8.5	8.4	8.3	8.5		
10	0.6		7.4	8.2	8.3	8.3	8.3	8.2	8.3	8.3	8.3	8.4	8.6	8.7	8.7	8.4	8.7	7.3	8.9
44	1.1	J10265	7.4	8.1	7.8	8.1	8.1	8.1	8.1	8.1	8.2	8.3	8.8	8.6	8.7	8.2	8.7		
25	1.2	A9-1	7.4	8.0	8.0	8.0	7.9	7.9	7.9	8.0	8.0	8.1	8.5	8.5	8.6	8.2	8.5		
1	1.3		7.0	8.2	8.2	7.6	7.9	8.0	7.9	8.1	7.9	7.7	8.2	8.3	8.0	8.0	8.0		
9	1.4		7.3	8.2	8.1	8.1	8.1	8.0	8.1	8.0	8.1	8.4	8.7	8.8	8.4	8.4	8.7		
23	1.5		7.3	8.0	8.1	8.1	8.1	7.9	8.0	8.0	8.1	8.2	8.6	8.6	8.5	8.3	8.6		
24	1.6		7.3	7.9	8.0	8.0	8.0	7.9	7.9	8.0	8.0	8.2	8.6	8.6	8.6	8.2	8.5	7.0	8.8
48	2.1	J10264	7.5	8.0	8.1	8.1	7.8	8.0	8.0	8.0	8.0	8.2	8.5	8.7	9.0	8.5	8.8		
33	2.2	A3-1	7.5	7.9	8.1	8.1	8.0	7.9	7.9	7.9	8.0	8.1	8.5	8.4	8.2	8.1	8.5		
19	2.3		7.3	8.0	7.8	8.0	8.0	7.9	7.9	7.9	7.9	8.1	8.5	8.5	8.4	8.2	8.5		
2	2.4		7.3	8.1	8.2	8.1	8.2	8.0	7.5	8.1	8.1	8.1	8.2	8.4	8.4	8.2	8.2		
22	2.5		7.4	8.1	8.0	8.1	8.1	8.0	8.1	8.0	8.0	8.2	8.7	8.8	8.3	8.2	8.6		
18	2.6		7.3	8.1	7.8	8.1	8.0	7.9	7.9	8.0	7.9	8.2	8.6	8.7	8.7	8.3	8.6	7.3	9.0
46	3.1	J10262	7.6	8.2	8.1	8.2	8.0	8.1	8.2	8.1	8.2	8.4	8.7	8.8	9.1	8.5	8.8		
15	3.2	A6-2	7.4	8.2	8.1	8.2	8.1	8.0	7.9	8.1	8.1	8.2	8.5	8.6	8.6	8.4	8.6		
47	3.3		7.5	8.1	8.1	8.2	7.9	8.0	8.1	8.1	8.0	8.3	8.6	8.8	8.9	8.4	8.8		
35	3.4		7.4	8.1	8.0	8.1	8.0	7.9	8.0	8.0	8.0	8.1	8.3	8.5	8.6	8.2	8.5		
16	3.5		7.7	8.2	8.0	8.2	8.1	8.0	7.9	8.1	8.1	8.2	8.7	8.9	8.8	8.4	8.7		
38	3.6		7.3	8.2	8.1	8.2	8.1	8.0	8.1	8.1	8.1	8.1	8.6	8.8	8.8	8.4	8.5	7.3	9.1
8	4.1	J10497	7.6	8.2	8.3	8.3	8.2	8.2	8.3	8.3	8.3	8.3	8.6	8.9	8.4	8.7	8.6		
39	4.2	A5-2	7.5	8.1	8.3	8.3	8.2	8.1	8.2	8.2	8.3	8.2	8.6	8.9	9.0	8.5	8.7		
21	4.3		7.7	8.1	8.2	8.2	8.2	8.2	8.3	8.3	8.4	8.2	8.4	8.6	8.8	8.5	8.5		
40	4.4		7.6	8.1	8.1	8.3	8.2	8.2	8.3	8.3	8.3	8.3	8.6	8.8	9.0	8.4	8.7		
45	4.5		7.5	8.1	8.2	8.3	8.1	8.1	8.3	8.3	8.3	8.4	8.8	8.7	9.0	8.5	8.7		
11	4.6		7.6	8.1	8.2	8.3	8.3	8.2	8.3	8.4	8.5	8.5	8.6	8.6	9.1	8.6	8.8	7.5	9.1
32	5.1	J10494	7.5	8.0	8.1	8.2	8.2	8.1	8.2	8.2	8.2	8.2	8.6	8.5	8.6	8.2	8.6		
20	5.2	A4-1	7.4	8.0	8.1	8.2	8.1	8.0	8.1	8.1	8.1	8.1	8.3	8.4	8.0	8.2	8.5		
42	5.3		7.3	8.2	8.2	8.1	8.1	8.1	8.2	8.1	8.2	8.4	8.6	8.6	8.7	8.2	8.7		
14	5.4		7.4	8.1	8.0	8.1	8.1	8.0	7.7	8.1	8.0	8.2	8.6	8.7	8.2	8.2	8.6		
3	5.5		7.4	8.3	8.2	8.3	8.2	8.0	8.0	8.2	8.2	8.2	8.3	8.3	8.3	8.2	8.4		
31	5.6		7.5	8.1	8.0	8.2	8.1	8.1	8.2	8.1	8.1	8.2	8.6	8.5	8.7	8.2	8.7	7.3	8.7
7	6.1	J10493	7.3	8.1	8.1	8.1	8.0	8.0	8.1	8.1	8.1	8.2	8.5	8.4	8.3	8.4	8.5		
5	6.2	A2-2	7.4	8.2	8.2	8.1	8.1	8.0	8.1	8.2	8.2	8.2	8.4	8.3	8.3	8.2	8.4		
29	6.3		7.4	8.0	7.9	8.0	7.9	7.9	8.0	8.0	7.9	7.5	8.4	8.5	7.7	8.2	8.5		
28	6.4		7.4	8.0	8.1	8.2	8.1	8.1	8.1	8.1	8.0	8.2	8.5	8.6	8.7	8.2	8.6		
17	6.5		7.5	8.1	8.1	8.2	8.2	8.1	8.2	8.3	8.2	8.3	8.6	8.6	8.6	8.4	8.7		
12	6.6		7.4	8.2	8.0	8.2	8.1	8.1	8.1	8.2	8.3	8.5	8.7	8.9	8.1	8.4	8.7	7.3	8.9
6	7.1	J10486	7.2	8.1	8.1	8.2	8.1	8.0	8.1	8.1	8.1	8.3	8.5	8.6	8.6	8.2	8.5		
37	7.2	A1-1	7.2	8.0	8.1	8.1	8.1	7.9	8.0	8.0	8.0	8.1	8.4	8.5	8.1	8.1	8.5		
4	7.3		7.4	8.2	8.1	8.1	8.1	7.9	7.9	8.1	8.0	8.2	8.5	8.4	8.3	8.1	8.4		
34	7.4		7.3	8.1	8.0	8.1	8.0	7.9	8.0	8.0	8.0	8.1	8.3	8.3	8.3	8.1	8.4		
36	7.5		7.2	8.1	8.0	8.1	8.0	7.9	8.0	8.1	8.1	8.1	8.4	8.5	8.7	8.3	8.5		
13	7.6		7.4	8.1	7.8	8.1	8.0	7.8	7.9	8.1	8.1	8.3	8.7	8.7	8.5	8.5	8.7	7.2	8.7
															Ranges		7.0	9.1

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JN
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Table 7

AQUA SURVEY, INC.
14-DAY SOLID PHASE READINGS

CLIENT: Rov F. Weston   TEST START DATE: 6/25/97   PARAMETER: Total NH3 (mg/L)
JOB #: 97-286   ORGANISM: *H. azteca*

Position #	ID#	Sample	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Low	High
43	0.1	Control	1.36														ND		
30	0.2		ND														ND		
27	0.3		ND														ND		
41	0.4		ND														ND		
26	0.5		ND														ND		
10	0.6		ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.36
44	1.1	J10265	0.46														ND		
25	1.2	A9-1	0.55														ND		
1	1.3		0.46	0.29	0.39	0.24	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.24	ND		
9	1.4		0.49														ND		
23	1.5		0.54														ND		
24	1.6		0.55														ND	ND	0.55
48	2.1	J10264	0.92														ND		
33	2.2	A3-1	0.90														ND		
19	2.3		0.91														ND		
2	2.4		0.83	1.60	2.72	3.01	3.26	2.89	2.60	0.82	0.13	ND	ND	ND	ND	ND	ND		
22	2.5		0.90														ND		
18	2.6		0.87														ND	ND	3.26
46	3.1	J10262	1.36														ND		
15	3.2	A6-2	1.23	1.78	2.55	2.22	1.44	0.43	ND	ND	ND	ND	ND	ND	ND	ND	ND		
47	3.3		1.40														ND		
35	3.4		1.33														ND		
16	3.5		1.30														ND		
38	3.6		1.34														ND	ND	2.55
8	4.1	J10497	ND	0.14	0.18	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		
39	4.2	A5-2	ND														ND		
21	4.3		ND														ND		
40	4.4		ND														ND		
45	4.5		ND														ND		
11	4.6		ND														ND	ND	0.18
32	5.1	J10494	ND														ND		
20	5.2	A4-1	ND														ND		
42	5.3		ND														ND		
14	5.4		ND														ND		
3	5.5		ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		
31	5.6		ND														ND	ND	ND
7	6.1	J10493	0.39														ND		
5	6.2	A2-2	0.50	0.70	1.01	0.48	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		
29	6.3		0.39														ND		
28	6.4		0.49														ND		
17	6.5		0.42														ND		
12	6.6		0.39														ND	ND	1.01
6	7.1	J10486	1.38														ND		
37	7.2	A1-1	1.43														ND		
4	7.3		1.35	2.13	2.80	2.38	1.19	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		
34	7.4		1.44														ND		
36	7.5		1.44														ND		
13	7.6		1.40														ND	ND	2.80

Ranges   ND   3.26

Raw Data

REAC, Edison, NJ
(908) 321-4200
EPA Contract 68-C4-0022

CHAIN OF CUSTODY RECORD

Project Name: Cornell Dubilier
Project Number: 03347-142-001-2273-01
RFW Contact: Scott Grossman Phone (908) 321-4200

No: 05339

SHEET NO. 1 OF 1

Sample Identification

Analyses Requested

REAC #	Sample No.	Sampling Location	Matrix	Date Collected	# of Bottles	Container/Preservative	Tox Test				
*	J10265	A9-1	Sd	6-20-97	2	32oz glass/4°C	X				
	J10264	A3-1		6-20-97							
	J10262	A6-2		6-19-97							
	J10497	A5-2		6-19-97							
	J10494	A4-1									
	J10493	A2-2									
	J10486	A1-1		6-18-97							

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Matrix:

SD -	Sediment	PW -	Potable Water	S -	Soil
DS -	Drum Solids	GW -	Groundwater	W -	Water
DL -	Drum Liquids	SW -	Surface Water	O -	Oil
X -	Other	SL -	Sludge	A -	Air

Special Instructions:

cooler #2328

Note: Sediment for tox test Hyalella azteca

QC: Cen

FOR SUBCONTRACTING USE ONLY

FROM CHAIN OF CUSTODY #

Items/Reason	Relinquished By	Date	Received By	Date	Time	Items/Reason	Relinquished By	Date	Received By	Date	Time
All Analysis	Jennifer Korp	6/20/97	A. Hornberger	6/21/97	10:30						

FORM #4

8/94

Received @ 13°C

ASI, INC.
SAMPLE RECEIVING FORM

Client:	Weston	Reac			97-286			
Shipped Via: Fed. Ex					**# of Shipping Containers:** 1			
Type of Shipping Container Coder		**Custody Seal** Present ✓ Absent _ Broken _		**Condition of Shipping Containers:** Acceptable ✓ Unacceptable ___				
ASI #	**Sample ID**	**Type of Container**	**Number of Containers**	**Condition of Samples†**	**Temp. °C**	**Ice +**	**Type of Sample***	
1. 71164	J10265 A9-1	G/1 l	2	A	13°C	cold water	fresh water sed.	
2. 71165	J10264 A3-1		2	A				
3. 71166	J10262 A6-2		2	A				
4. 71167	J10497 A5-2		2	A				
5. 71168	J10494 A4-1		2	A				
6. 71169	J10493 A2-2		2	A				
7. 71170	J10486 A1-1	↓	2	A	↓	↓	↓	
8.								
9.								
10.								

Notes: (Discrepancies Between Sample Label and COC Record)

Opened /Received by:	J. Hombergen		Date/Time: 6/21 10:30

S - Soil
Se - Sediment
Sl - Sludge
W - Water
E - Effluent

A - Acceptable
U - Unusable or Contaminated

I - Ice
D - Dry Ice
B - Blue Ice
N - None

7/96

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Job # 97-286, H. azteca 14 day solid phase test

Position	Sample	ASI No.	
43	Control	71174	0.1
30		*	0.2
27		*	0.3
41		*	0.4
26		*	0.5
10		*	0.6
44	J10265	71164	1.1
25	A9-1	*	1.2
1		*	1.3
9		*	1.4
23		*	1.5
24		*	1.6
48	J10264	71165	2.1
33	A3-1	*	2.2
19		*	2.3
2		*	2.4
22		*	2.5
18		*	2.6
46	J10262	71166	3.1
15	A6-2	*	3.2
47		*	3.3
35		*	3.4
16		*	3.5
38		*	3.6
8	J10497	71167	4.1
39	A5-2	*	4.2
21		*	4.3
40		*	4.4
45		*	4.5
11		*	4.6
32	J10494	71168	5.1
20	A4-1	*	5.2
42		*	5.3
14		*	5.4
3		*	5.5
31		*	5.6
7	J10493	71169	6.1
5	A2-2	*	6.2
29		*	6.3
28		*	6.4
17		*	6.5
12		*	6.6
6	J10486	71170	7.1
37	A1-1		7.2
4			7.3
34			7.4
36			7.5
13			7.6

97-286   14-day Solid Phase

Aqua Survey, Inc.
Solid Phase Readings

Client: __RFW__   Test Start Date: __6/25/97__   Parameter: __Counts__

Job #: __97-296__   Test End Date: __7/9/97__   Organism: __H. azteca__

Beaker	Initial Count	Final Count	Beaker	Initial Count	Final Count	Notes
1	20	19	25	20	20	
2	20	17	26	20	16	
3	20	17	27	20	17	
4	20	18	28	20	19	
5	20	17	29	20	20	
6	20	17	30	20	17	
7	20	19	31	20	16	
8	20	17	32	20	20	
9	20	16	33	20	14	
10	20	18	34	20	17	
11	20	18	35	20	17	
12	20	19	36	20	19	
13	20	20	37	20	19	
14	20	20	38	20	18	
15	20	17	39	20	15	
16	20	17	40	20	18	
17	20	19	41	20	16	
18	20	13	42	20	17	
19	20	14	43	20	16	
20	20	19	44	20	19	
21	20	11	45	20	20	
22	20	17	46	20	20	
23	20	18	47	20	17	
24	20	18	48	20	17	
Date	6/25/97	7/9/97	Date	6/25/97	7/9/97	
Initials	CAN	CAN	Initials	CAN	CAN	

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Client: RFW/RFAC   Test Start Date: 6/25/97   Parameter: Alkalinity, Hardness, Con

Job #: 97-286   Test End Date: 7/9/97   Organism: H. azteca

Sample ID #	Initial			Final		
	Alkalinity	Hardness	Conductivity	Alkalinity	Hardness	Conductivity
7117⁻	196	112	530	260	160	620
71164	180	116	520	196	124	540
I1165	224	124	550	208	140	550
71166	196	124	530	196	120	530
31167	204	124	527	248	156	600
71168	188	136	532	200	132	545
71169	216	156	557	260	164	580
71170	204	140	547	196	112	520
Date	6/25/97	6/25/97	6/25/97	7/9/97	7/9/97	7/9/97
Initials	CAN	CAN	AN	KF	KF	KF.

Notes: Intial Parameters read. Some D.O. levels were low
           _____ communicates prior to starting test on 6/25/97 AN

           Sample 71170 (JIC486) had tube-dwelling worms present on
Surface after serving. AN 6/25/97
           Sample 71165 Same as above AN

AQUA SURVEY, INC.
SOLID PHASE

Client: REIN REAC   Test Start Date: 6/25/97

Job #: 97-286   Organism: H azteca   Water: Well

Date	Day	Volume	1st Exchange		2nd Exchange		3rd Exchange		4th Exchange	
			Time	Initials	Time	Initials	Time	Initials	Time	Initials
6/25	0	200mL	N/A	AN						
6/26	1		N/A	AN						
6/27	2	200mL	7:00P	AH						
6/28	3		NA	NA						
6/29	4	200mL	12:-	AH						
6/30	5									
7/1	6	200mL	6:37 PM	AH			N/A			
7/2	7									
7/3	8	200mL	4:00	AN						
7/4	9									
7/5	10	200mL	1030	AN						
7/6	11									
7/7	12	200mL	3 pm	TD						
7/8	13									
7/9	14	200mL	Na							

Notes:

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Aqua Survey, Inc.
Solid Phase Readings

Client: _REW RFAC_ Test Start Date: _6/25/07_ Parameter: _Ammonia_

Job #: _07-296_ Organism: _H. azteca_

Beaker	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Notes
1	0.46	0.29	0.39	0.24	NT	ND	ND	ND	NT	ND	ND	ND	ND	0.24	ND	
2	0.83	1.60	2.72	3.01	3.26	2.89	2.60	0.82	0.13	ND	ND	ND	ND	ND	ND	
3	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
4	1.35	2.13	2.80	2.38	1.19	ND	NT	ND	ND	ND	ND	ND	ND	ND	ND	
5	0.56	0.70	1.01	0.48	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
6	1.58														ND	
7	0.39														ND	
8		0.04	0.18	ND							ND	ND	ND	ND	ND	
9	0.44														ND	
10	ND	ND	ND	ND	NT	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
11	N/D														ND	
12	0.39														ND	
13	1.40														ND	
14	ND														ND	
15	1.23	1.78	2.55	2.22	1.44	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
16	1.30														ND	
17	0.42														ND	
18	0.87														ND	
19	0.91														ND	
20	ND														ND	
21	ND														ND	
22	0.90														ND	
23	0.54														ND	
24	0.55														ND	
Date	6/25	6/26	6/27	6/28	6/29	6/30	7/1	7/2	7/3	7/4	7/5	7/6	7/7	7/8	7/9	
Initials																

Aqua Survey, Inc.
Solid Phase Readings

Client: KTW REAC   Test Start Date: 6/25/97   Parameter: Ammonia

Job #: 97-286   Organism: H. azteca

Beaker	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Notes
25	0.55														ND	
26	ND														ND	
27	ND														ND	
28	0.49														ND	
29	0.39														ND	
30	ND														ND	
31	ND														ND	
32	ND														ND	
33	ND														ND	
34	1.44														ND	
35	1.33														ND	
36	1.44														ND	
37	1.43														ND	
38	1.34														ND	
39	ND														ND	
40	ND														ND	
41	ND														ND	
42	ND														ND	
43	1.36														ND	
44	0.46														ND	
45	ND														ND	
46	1.36														ND	
47	1.40														ND	
48	0.92														ND	
Date	6/25														7/9	
Initials	LH														R	

Aqua Survey, Inc.
Solid Phase Readings

Client: REV REAC Test Start Date: 6/25/97 Parameter: Observations

Job #: 97-296 Organism: H. nztera

Key: D = Dead S = Surface N = Nothing Unusual

Beaker	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Notes
1	②	N	N	N①	N	N	N	N②	1S	5S②	6S	4S	5S	4S	4S	
2	①	2S	2S	N①	N	N	N	N②	N②	1S②	1S	N	7S	3S	N	
3	②	2S	N	N	N	N	N	N	1S	1S	N	N	3S	3S	2S	
4	①	N	N	N	N	N	N	N②	2S	3S②	2S	3S	5S	4S	2S	
5	②	N	N	N	N	N	N	N	N	N	N	N	2S	N	2S	
6	①	N	N	N①	N	N	N	N②	2S②	2S②	3S	1S	4S	1S	1S	
7	②	N	N	N①	N	N	3S	N②	2S	2S	2S	2S	5S	1S	N	
8	②	N	N	N	N		N	1S	1S	1S	2S	1S	2S	1S		
9	②	N	1S	1S	N	N	N	N	1S	2S②	2S	3S	2S	4S	3S	
10	②	N	N	N	N	N	N	N	2S	2S	3S	4S	4S	4S	N	
11	②	N	N	N	N	N	1S	N	2S	N	3S	1S	2S	3S	N	
12	②	N	1S	N	N	N	N	N②	N	N②	N	1S	4S	2S	2S	
13	①	N	N① N	N②	N	N	N	N②	2S	N②	N	N	N	N	2S	
14	②	N	1S	N	1S	S	N	N	2S	N	N	N	1S	N	2S	
15	②	N	2S	N	N	N	N	N	N	N②	1S	2S	N	N	N	
16	②	N	N	N	N	N	N	N	N	1S	2S	3S	3S	1S	3S	
17	②	N	N	N	N	N	N	N②	N	1S	2S	N	N	N	2S	
18	①	1S	N① N	N	N	N	N	N②	4S	5S②	4S	N	3S	2S	1S	
19	①	1S	N③ 3S	N	N	2S	N	N②	5S	4S②	6S	5S	5S	3S	1S	
20	②	N	N	N	N	1S	N	N	1S	3S	2S	3S	5S	3S	1S	
21	②	N	N	N	N	N	N	N	N	N	1S	1S	N	2S	2S	
22	①	N	N① 4S	N①	N	N	N②	N② N②	1S	N	1S	2S	2S		
23	②	N① N	N	N	N	3S	N②	2S	N②	N	1S	2S	N	N	
24	②	N	N	N①	2S	N	1S	N②	1S	2S②	1S	N	4S	3S	N	
Date	6/25	6/26	6/27	6/29	6/29	6/30	7/1	7/2	7/3	7/4	7/5	7/6	7/7	7/8	7/9	
Initials	CAN	AN/TF	AN/KH	AH	AH	AH	SH	AN	AN/SH	TF	CAN	CAN	TF	AN	AN	

① tube-dwelling worms on sediment surface
② some organisms on surface, toted down in bullet water in Aqua

Aqua Survey, Inc.
Solid Phase Readings

Client: RFW REAC   Test Start Date: 6/25/97   Parameter: Observations

Job #: 97-296   Organism: H.Uztica

Key: D = Dead   S = Surface   N = Nothing Unusual

Beaker	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Notes
25	②	N	N	N	N⁰	N¹	N¹	N⁰	25	45⁰	35	45	15	25	N	
26	②	N	N	N	N	N	1S	N	1S	15	1S	N	25	N	N	
27	②	N	N	N	N	N	N	N	1S	15	N	N	25	N	N	
28	②	N	N	N⁰	N⁰	N¹	1S¹	N	N	15⁰	1S	25	15	N	1S	
29	②	N	N	N	N	N	N	25	15	15⁰	35	25	65	55	N	
30	②	N	N	N	N	N	N	N	1S	45	55	35	95	65	35	
31	②	1S	N	N	1S	45	N	35	15	N	25	N	35	35	25	
32	②	N	N	N	1S	N	N	N	35	25	N	35	45	25	25	
33	①	N⓪ N	N⓪	N¹	N	1S	N⓪	35	35⓪	25	15	45	25	35	
34	①	N⓪ N	N⓪	N¹	N	1S¹	25	35	35⓪	25	1S	45	25	25	
35	②	N	1S	N	N	1S	1S	N	N	N	35	N	N	35	25	
36	①	N⓪N	N⓪	N¹	N¹	1S¹	1S⓪	1S	1S⓪	1S	1S	25	35	1S	
37	①	N⓪N	N⓪	N¹	1S¹	1S¹	1S⓪	45	35⓪	35	25	65	N	1S	
38	②	N	N	N	N¹	N	N	N	N	N	1S	25	65	35	
39	②	N	N	N	N	1S	1S	1S	N	N	N	N	25	65	1S	
40	②	N	1S	N	N	N	2S	N	N	N	N	N	1S	25	1S	
41	②	N	N	N	1S	N	N	N	N	1S	N	N	N	N	25	
42	②	1S	N	N	25	35	25	35	55	65	45	35	N	N	35	
43	②	N	N	N	N	N	35	25	35	15	1S	35	1S	N	45	
44	②	N⓪N	N⓪	N¹	1S³	3S¹	25⓪	55	45	55	65	65	45	25	
45	②	N	N	N	N	N	1S	1S	35	45	45	25	1S	35	35	
46	②	N	N	N	N	N	N	N	N	N	1S	N	N	25	25	
47	②	N	N	N	N¹	N¹	N	N	N	N	N	N	25	1S	1S	
48	①	N⓪N	N⓪	N¹	N¹	1S¹	N⓪	33	35	35	25	35	25	1S	
Date	6/25	6/26	6/27	6/28	6/30	6/30	7/1	7/2	7/3	7/4	7/5	7/6	7/7	7/8	7/9	
Initials	CAN	AN/TD	AN/SH	NH	NH	NH	CAN	AN	T	CAN	CAN	T	CAN	CAN		

① tube-dwelling worms on surface   20
② some organisms on surface: turned down ...observations...

Aqua Survey, Inc.
Solid Phase Readings

Client: PFW REAC   Test Start Date: 6/25/97   Parameter: Feeding

Job #: 97-286   Organism: H. ayteca

Beaker	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Notes
1	No	No	Yes	No	No	No	No	Yes	No	No	Yes	No	No	Yes	No	
2																
3																
4																
5																
6																
7																
8																
9																
10																
11																
12																
13																
14																
15																
16																
17																
18																
19																
20																
21																
22																
23																
24	↓		↓							↓			↓	↓	↓	
Date	6/25	6/26	6/27	6/28	6/29	6/30	7/1	7/2	7/3	7/4	7/5	7/6	7/7	7/8	7/9	
Initials	CN	CN	SA CN	CN	CN	CN	SA	CN	TD	CN	CN	TD	TD	MA		

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Aqua Survey, Inc.
Solid Phase Readings

Client: _REU/REAC_   Test Start Date: _____   Parameter: _Folding_

Job #: _97-286_   Organism: _H. Azteca_

Beaker	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Notes
25	No	NO	YES							NO			NO	Yes	Nn	
26																
27																
28																
29																
30																
31																
32																
33																
34																
35																
36																
37																
38																
39																
40																
41																
42																
43																
44																
45																
46																
47																
48	↓		↓							↓			↓	↓	↓	
Date	6/25	6/26	6/27							TO			TO	TO	MF	
Initials	CAN	dAN	2M2							7/4			7/7	7/8	7/9	

YSI 6000 Time Series Report   Page 1

Date mm/dd/yy	Time hh:mm:ss	Temp C	SpCond uS/cm	Salinity PPT	DO mg/L	pH
6/25/97	14:03:25 /	23.86	502.00	0.2	6.03	6.97
6/25/97	14:05:43	23.85	545.00	0.3	4.46	7.29
6/25/97	14:06:23	23.94	523.00	0.2	5.99	7.43
6/25/97	14:07:09	23.97	527.00	0.2	6.08	7.37
6/25/97	14:07:55	23.97	555.00	0.3	5.94	7.35
6/25/97	14:08:49	23.96	532.00	0.3	6.50	7.21
6/25/97	14:10:42	23.96	546.00	0.3	5.92	7.30
6/25/97	14:11:27	23.96	518.00	0.2	5.96	7.58
6/25/97	14:12:11	23.99	507.00	0.2	6.03	7.32
6/25/97	14:13:14	23.91	519.00	0.2	6.93	7.38
6/25/97	14:13:52	23.99	520.00	0.2	6.33	7.62
6/25/97	14:14:36	23.98	557.00	0.3	5.99	7.40
6/25/97	14:15:07	23.98	577.00	0.3	5.97	7.40
6/25/97	14:15:25	23.99	506.00	0.2	6.76	7.42
6/25/97	14:15:44	23.99	527.00	0.2	7.12	7.38
6/25/97	14:16:15	23.98	527.00	0.2	6.60	7.71
6/25/97	14:16:35	23.99	550.00	0.3	7.26	7.47
6/25/97	14:16:56	24.00	547.00	0.3	6.89	7.30
6/25/97	14:17:53	23.98	550.00	0.3	4.15	7.31
6/25/97	14:18:24	23.97	556.00	0.3	5.79	7.43
6/25/97	14:18:56	23.97	527.00	0.2	6.48	7.65
6/25/97	14:19:28	23.98	564.00	0.3	4.98	7.42
6/25/97	14:20:02	23.96	505.00	0.2	5.83	7.30
6/25/97	14:20:26	23.91	508.00	0.2	6.12	7.32
6/25/97	14:20:54	23.97	501.00	0.2	6.07	7.37
6/25/97	14:21:33	23.97	517.00	0.2	6.53	7.49
6/25/97	14:22:01	24.00	518.00	0.2	6.44	7.55
6/25/97	14:23:04	23.97	554.00	0.3	6.05	7.39
6/25/97	14:23:41	23.95	577.00	0.3	5.94	7.38
6/25/97	14:24:11	24.00	517.00	0.2	6.60	7.55
6/25/97	14:24:39	24.00	510.00	0.2	6.69	7.53
6/25/97	14:25:10	23.99	506.00	0.2	6.51	7.46
6/25/97	14:25:39	24.00	544.00	0.3	5.58	7.45
6/25/97	14:26:38	23.95	537.00	0.3	5.96	7.25
6/25/97	14:28:22	23.87	527.00	0.2	6.02	7.43
6/25/97	14:28:51	23.86	558.00	0.3	5.83	7.20
6/25/97	14:29:16	23.98	529.00	0.2	6.22	7.18
6/25/97	14:29:34	24.00	526.00	0.2	6.47	7.28
6/25/97	14:30:07	23.89	519.00	0.2	5.97	7.45
6/25/97	14:30:49	23.95	529.00	0.2	6.22	7.59
6/25/97	14:31:21	23.95	519.00	0.2	6.23	7.49
6/25/97	14:31:52	23.90	507.00	0.2	6.25	7.32
6/25/97	14:32:17	23.89	521.00	0.2	6.40	7.32
6/25/97	14:32:40	23.67	533.00	0.3	6.92	7.42
6/25/97	14:32:59	23.74	528.00	0.2	6.79	7.45
6/25/97	14:33:29	23.87	527.00	0.2	6.57	7.56
6/25/97	14:33:46	24.00	529.00	0.3	6.81	7.45
6/25/97	14:34:05	23.90	545.00	0.3	6.03	7.50

YSI 6000 Time Series Report

Date mm/dd/yy	Time hh:mm:ss	Temp C	SpCond uS/cm	Salinity PPT	DO mg/L	pH
6/26/97	12:46:49 /	22.64	508.00	0.2	7.80	8.15
6/26/97	12:56:26	22.26	560.00	0.3	7.52	8.13
6/26/97	13:01:51	22.22	535.00	0.3	7.58	8.29
6/26/97	13:02:29	22.69	528.00	0.2	7.67	8.22
6/26/97	13:03:04	22.66	572.00	0.3	7.68	8.17
6/26/97	13:03:18	22.60	546.00	0.3	7.69	8.08
6/26/97	13:03:34	22.67	582.00	0.3	7.67	8.10
6/26/97	13:03:48	22.80	537.00	0.3	7.64	8.19
6/26/97	13:04:03	22.73	512.00	0.2	7.66	8.15
6/26/97	13:04:24	22.46	530.00	0.3	7.73	8.23
6/26/97	13:04:41	22.62	535.00	0.3	7.72	8.13
6/26/97	13:05:06	22.78	580.00	0.3	7.69	8.16
6/26/97	13:05:28	22.51	600.00	0.3	7.73	8.08
6/26/97	13:05:42	22.63	517.00	0.2	7.70	8.07
6/26/97	13:06:04	22.68	537.00	0.3	7.70	8.17
6/26/97	13:06:33	22.74	539.00	0.3	7.67	8.16
6/26/97	13:06:53	22.69	573.00	0.3	7.66	8.06
6/26/97	13:07:08	22.68	569.00	0.3	7.67	8.11
6/26/97	13:07:26	22.78	575.00	0.3	7.66	8.02
6/26/97	13:07:39	22.86	562.00	0.3	7.61	8.02
6/26/97	13:07:54	22.72	548.00	0.3	7.62	8.07
6/26/97	13:08:08	22.70	586.00	0.3	7.63	8.08
6/26/97	13:08:22	22.78	512.00	0.2	7.63	8.03
6/26/97	13:08:35	22.61	512.00	0.2	7.67	7.94
6/26/97	13:08:59	22.67	502.00	0.2	7.69	8.02
6/26/97	13:09:12	22.60	535.00	0.3	7.71	7.91
6/26/97	13:12:08	22.50	528.00	0.2	7.89	8.17
6/26/97	13:12:23	22.68	587.00	0.3	7.77	8.03
6/26/97	13:12:36	22.79	604.00	0.3	7.71	8.04
6/26/97	13:12:50	22.71	533.00	0.3	7.70	8.04
6/26/97	13:13:07	22.68	519.00	0.2	7.71	8.13
6/26/97	13:13:22	22.74	515.00	0.2	7.69	8.00
6/26/97	13:13:34	22.71	563.00	0.3	7.69	7.94
6/26/97	13:14:30	22.61	548.00	0.3	7.77	8.06
6/26/97	13:14:50	22.69	537.00	0.3	7.64	8.07
6/26/97	13:15:04	22.65	560.00	0.3	7.63	8.07
6/26/97	13:15:19	22.68	533.00	0.3	7.63	8.02
6/26/97	13:15:39	22.75	539.00	0.3	7.63	8.15
6/26/97	13:15:56	22.66	530.00	0.3	7.66	8.09
6/26/97	13:16:11	22.63	543.00	0.3	7.69	8.14
6/26/97	13:16:25	22.70	532.00	0.3	7.67	8.13
6/26/97	13:16:46	22.70	506.00	0.2	7.67	8.15
6/26/97	13:17:14	22.73	537.00	0.3	7.68	8.21
6/26/97	13:17:30	22.69	534.00	0.3	7.66	8.08
6/26/97	13:17:43	22.69	538.00	0.3	7.66	8.06
6/26/97	13:17:59	22.76	533.00	0.3	7.66	8.17
6/26/97	13:18:09	22.83	530.00	0.3	7.65	8.06
6/26/97	13:18:20	22.73	564.00	0.3	7.66	8.00

Date mm/dd/yy	Time hh:mm:ss	Temperature C	DO mg/L	pH
6/27/97	9:47:06	22.59	7.81	8.24
6/27/97	9:47:37	22.57	7.70	8.16
6/27/97	9:48:10	22.61	7.56	8.23
6/27/97	9:49:26	22.62	7.67	8.12
6/27/97	9:50:07	22.70	7.58	8.24
6/27/97	9:50:42	22.67	7.57	8.13
6/27/97	9:51:16	22.72	7.49	8.08
6/27/97	9:52:27	22.65	7.66	8.27
6/27/97	9:53:00	22.77	7.61	8.05
6/27/97	9:53:35	22.67	7.55	8.30
6/27/97	9:54:10	22.70	7.52	8.24
6/27/97	9:55:31	22.67	7.63	8.01
6/27/97	9:58:31	22.65	6.88	7.77
6/27/97	9:59:05	22.67	7.16	8.04
6/27/97	10:00:05	22.79	7.45	8.13
6/27/97	10:01:09	22.69	7.55	8.01
6/27/97	10:01:42	22.76	7.37	8.12
6/27/97	10:02:18	22.76	6.39	7.78
6/27/97	10:03:06	22.84	6.61	7.84
6/27/97	10:04:29	22.76	7.71	8.08
6/27/97	10:05:11	22.80	7.66	8.22
6/27/97	10:05:36	22.72	7.60	8.03
6/27/97	10:06:34	22.77	7.56	8.07
6/27/97	10:08:15	22.76	7.72	7.95
6/27/97	10:08:49	22.71	7.66	7.97
6/27/97	10:09:21	22.78	7.45	8.08
6/27/97	10:09:50	22.88	7.19	8.21
6/27/97	10:10:48	22.81	7.53	8.05
6/27/97	10:12:44	22.75	7.59	7.93
6/27/97	10:13:13	22.72	7.46	8.26
6/27/97	10:14:01	22.79	7.54	8.03
6/27/97	10:14:31	22.72	7.56	8.12
6/27/97	10:15:36	22.75	7.65	8.09
6/27/97	10:16:07	22.89	7.55	7.98
6/27/97	10:16:38	22.77	7.41	7.98
6/27/97	10:17:10	22.76	7.23	8.02
6/27/97	10:18:29	22.73	7.62	8.08
6/27/97	10:19:06	22.83	7.61	8.11
6/27/97	10:19:39	22.77	7.60	8.26
6/27/97	10:19:59	22.79	7.60	8.09
6/27/97	10:21:02	22.75	7.62	8.24
6/27/97	10:21:31	22.81	7.63	8.15
6/27/97	10:22:02	22.64	7.67	8.14
6/27/97	10:22:34	22.73	7.52	7.83
6/27/97	10:23:43	22.74	7.60	8.19
6/27/97	10:24:22	22.76	7.62	8.14
6/27/97	10:24:51	22.70	7.64	8.10
6/27/97	10:25:34	22.86	7.53	8.09

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Date mm/dd/yy	Time hh:mm:ss	Temperature C	DO mg/L	pH
6/28/97	10:07:03	22.50	7.79	7.57
6/28/97	10:07:39	22.40	7.71	8.11
6/28/97	10:08:06	22.31	7.65	8.25
6/28/97	10:09:22	22.50	7.65	8.10
6/28/97	10:09:51	22.42	7.62	8.14
6/28/97	10:10:28	22.53	7.55	8.20
6/28/97	10:10:53	22.52	7.52	8.09
6/28/97	10:14:09	22.52	7.36	8.25
6/28/97	10:14:32	22.50	7.40	8.09
6/28/97	10:15:01	22.60	7.43	8.32
6/28/97	10:15:28	22.60	7.47	8.28
6/28/97	10:16:35	22.60	7.60	8.17
6/28/97	10:17:08	22.70	7.53	8.06
6/28/97	10:17:31	22.60	7.48	8.12
6/28/97	10:17:57	22.61	7.47	8.15
6/28/97	10:19:05	22.66	7.58	8.18
6/28/97	10:19:38	22.67	7.57	8.20
6/28/97	10:20:09	22.67	7.53	8.11
6/28/97	10:20:35	22.70	7.45	8.03
6/28/97	10:21:38	22.60	7.57	8.20
6/28/97	10:22:01	22.62	7.60	8.22
6/28/97	10:22:30	22.70	7.56	8.13
6/28/97	10:23:02	22.80	7.49	8.08
6/28/97	10:24:02	22.75	7.54	7.99
6/28/97	10:24:29	22.70	7.50	8.04
6/28/97	10:25:22	22.69	7.55	8.25
6/28/97	10:25:54	22.79	7.50	8.27
6/28/97	10:26:25	22.80	7.47	8.15
6/28/97	10:27:44	22.80	7.49	8.00
6/28/97	10:28:11	22.61	7.46	8.30
6/28/97	10:29:11	22.83	7.56	8.15
6/28/97	10:29:42	22.65	7.62	8.23
6/28/97	10:30:40	22.76	7.61	8.12
6/28/97	10:31:13	22.76	7.51	8.09
6/28/97	10:31:45	22.76	7.45	8.12
6/28/97	10:32:12	22.70	7.45	8.13
6/28/97	10:33:11	22.67	7.57	8.10
6/28/97	10:33:38	22.76	7.55	8.18
6/28/97	10:34:08	22.70	7.57	8.26
6/28/97	10:34:17	22.72	7.58	8.25
6/28/97	10:35:12	22.71	7.69	8.35
6/28/97	10:35:40	22.76	7.67	8.14
6/28/97	10:36:04	22.56	7.69	8.33
6/28/97	10:36:33	22.66	7.64	8.11
6/28/97	10:37:33	22.67	7.66	8.26
6/28/97	10:38:04	22.66	7.65	8.20
6/28/97	10:38:31	22.61	7.64	8.23
6/28/97	10:38:59	22.70	7.59	8.10

YSI 6000 Time Series Report

Date mm/dd/yy	Time hh:mm:ss	Temperature C	DO mg/L	pH
6/29/97	10:48:21	22.53	8.08	7.91
6/29/97	10:48:49	22.57	7.98	8.20
6/29/97	10:49:09	22.60	7.91	8.22
6/29/97	10:49:53	22.63	7.88	8.05
6/29/97	10:50:10	22.68	7.82	8.09
6/29/97	10:50:56	22.78	7.81	8.12
6/29/97	10:51:11	22.80	7.79	8.04
6/29/97	10:51:26	22.76	7.77	8.20
6/29/97	10:51:51	22.82	7.73	8.09
6/29/97	10:52:48	22.80	7.58	8.34
6/29/97	10:53:00	22.72	7.62	8.26
6/29/97	10:53:16	22.72	7.64	8.10
6/29/97	10:53:42	22.80	7.61	7.97
6/29/97	10:54:33	22.83	7.47	8.06
6/29/97	10:55:01	22.90	7.54	8.10
6/29/97	10:55:17	22.80	7.61	8.09
6/29/97	10:55:38	22.80	7.67	8.15
6/29/97	10:56:28	22.88	7.57	8.04
6/29/97	10:56:52	22.90	7.50	8.01
6/29/97	10:57:06	22.79	7.49	8.09
6/29/97	10:57:31	22.87	7.55	8.22
6/29/97	10:58:15	22.94	7.55	8.06
6/29/97	10:58:32	22.87	7.52	8.05
6/29/97	10:58:56	22.87	7.57	7.97
6/29/97	10:59:15	22.84	7.60	7.94
6/29/97	10:59:54	22.93	7.54	8.23
6/29/97	11:00:22	23.02	7.50	8.27
6/29/97	11:00:45	22.86	7.60	8.11
6/29/97	11:02:13	22.90	7.40	7.92
6/29/97	11:02:32	22.79	7.40	8.28
6/29/97	11:02:56	22.85	7.48	8.13
6/29/97	11:03:40	22.84	7.71	8.21
6/29/97	11:04:32	22.83	7.66	8.02
6/29/97	11:05:03	22.96	7.52	7.99
6/29/97	11:05:26	22.82	7.52	8.02
6/29/97	11:05:50	22.85	7.54	8.02
6/29/97	11:06:43	22.80	7.52	8.08
6/29/97	11:07:03	22.80	7.55	8.14
6/29/97	11:07:25	22.79	7.62	8.18
6/29/97	11:07:39	22.74	7.70	8.16
6/29/97	11:08:37	22.80	7.66	8.34
6/29/97	11:08:58	22.81	7.70	8.12
6/29/97	11:09:16	22.63	7.78	8.12
6/29/97	11:09:44	22.79	7.78	8.07
6/29/97	11:10:37	22.77	7.75	8.05
6/29/97	11:11:04	22.80	7.71	7.97
6/29/97	11:11:51	22.70	7.76	7.88
6/29/97	11:12:21	22.88	7.61	7.80

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Date mm/dd/yy	Time hh:mm:ss	Temperature C	DO mg/L	pH
6/30/97	12:40:37	22.63	7.25	8.00
6/30/97	12:41:01	22.58	7.31	8.02
6/30/97	12:41:17	22.69	7.29	8.04
6/30/97	12:41:45	22.79	7.31	7.93
6/30/97	12:42:08	22.83	7.30	8.00
6/30/97	12:43:22	22.76	7.46	8.00
6/30/97	12:44:01	22.84	7.41	8.00
6/30/97	12:44:46	22.79	7.43	8.20
6/30/97	12:45:14	22.90	7.39	7.95
6/30/97	12:45:31	22.80	7.39	8.17
6/30/97	12:45:46	22.77	7.40	8.21
6/30/97	12:47:11	22.70	7.57	8.09
6/30/97	12:47:38	22.83	7.45	7.84
6/30/97	12:47:58	22.80	7.33	7.95
6/30/97	12:48:26	22.81	7.29	7.97
6/30/97	12:48:48	22.77	7.33	7.95
6/30/97	12:49:04	22.77	7.36	8.10
6/30/97	12:50:04	22.73	7.43	7.89
6/30/97	12:50:15	22.78	7.33	7.87
6/30/97	12:51:03	22.78	7.26	8.04
6/30/97	12:51:27	22.83	7.32	8.18
6/30/97	12:51:48	22.78	7.37	7.97
6/30/97	12:52:15	22.86	7.33	7.93
6/30/97	12:53:13	22.74	7.45	7.87
6/30/97	12:53:27	22.83	7.42	7.86
6/30/97	12:53:39	22.87	7.40	8.08
6/30/97	12:53:54	23.01	7.36	8.20
6/30/97	12:54:08	22.97	7.36	8.08
6/30/97	12:55:31	22.80	7.45	7.92
6/30/97	12:55:47	22.86	7.39	8.17
6/30/97	12:55:56	22.89	7.38	8.13
6/30/97	12:56:07	22.91	7.37	8.08
6/30/97	12:56:22	22.89	7.39	7.94
6/30/97	12:56:36	22.92	7.36	7.90
6/30/97	12:57:39	22.76	7.50	7.90
6/30/97	12:57:52	22.85	7.45	7.91
6/30/97	12:58:18	22.86	7.40	7.93
6/30/97	12:58:46	22.88	7.37	7.98
6/30/97	12:59:03	22.87	7.38	8.14
6/30/97	12:59:22	22.85	7.40	8.20
6/30/97	13:00:38	22.80	7.57	8.26
6/30/97	13:00:50	22.84	7.56	8.12
6/30/97	13:01:07	22.73	7.59	8.20
6/30/97	13:01:29	22.81	7.57	8.08
6/30/97	13:01:43	22.85	7.55	8.13
6/30/97	13:01:59	22.85	7.55	8.08
6/30/97	13:03:12	22.85	7.54	8.03
6/30/97	13:03:42	22.97	7.43	7.95

Date mm/dd/yy	Time hh:mm:ss	Temperature C	DO mg/L	pH
7/01/97	14:41:36	22.47	7.47	7.94
7/01/97	14:41:58	22.52	6.51	7.54
7/01/97	14:42:31	22.65	6.63	7.96
7/01/97	14:42:58	22.59	7.17	7.93
7/01/97	14:43:22	22.72	7.21	8.08
7/01/97	14:43:45	22.63	7.35	8.13
7/01/97	14:44:03	22.75	7.42	8.08
7/01/97	14:45:16	22.60	7.81	8.31
7/01/97	14:45:51	22.81	7.78	8.07
7/01/97	14:46:18	22.67	7.78	8.27
7/01/97	14:46:39	22.69	7.74	8.33
7/01/97	14:47:11	22.66	7.78	8.14
7/01/97	14:47:43	22.74	7.74	7.92
7/01/97	14:55:47	22.60	7.86	7.66
7/01/97	14:56:03	22.70	7.79	7.89
7/01/97	14:56:34	22.70	7.75	7.94
7/01/97	14:56:57	22.73	7.72	8.19
7/01/97	14:57:26	22.73	7.73	7.94
7/01/97	14:57:52	22.84	7.54	7.90
7/01/97	14:58:16	22.81	7.43	8.07
7/01/97	14:58:39	22.82	7.48	8.27
7/01/97	14:58:55	22.80	7.55	8.10
7/01/97	14:59:22	22.85	7.53	8.01
7/01/97	15:00:31	22.86	7.71	7.92
7/01/97	15:01:09	22.88	7.68	7.94
7/01/97	15:01:37	22.94	7.64	8.23
7/01/97	15:02:11	23.08	7.52	8.30
7/01/97	15:02:38	22.94	7.53	8.09
7/01/97	15:04:24	22.79	7.80	7.95
7/01/97	15:04:44	22.86	7.67	8.23
7/01/97	15:05:03	22.87	7.66	8.16
7/01/97	15:05:22	22.86	7.68	8.16
7/01/97	15:05:54	22.87	7.68	7.92
7/01/97	15:06:15	22.92	7.58	7.95
7/01/97	15:06:35	22.84	7.54	7.98
7/01/97	15:06:49	22.82	7.52	8.00
7/01/97	15:08:14	22.79	7.81	8.00
7/01/97	15:08:36	22.75	7.77	8.05
7/01/97	15:08:53	22.74	7.73	8.22
7/01/97	15:09:18	22.77	7.63	8.26
7/01/97	15:09:37	22.69	7.51	8.28
7/01/97	15:09:57	22.81	7.48	8.17
7/01/97	15:10:24	22.61	7.64	8.28
7/01/97	15:10:51	22.78	7.67	8.09
7/01/97	15:11:52	22.73	7.86	8.25
7/01/97	15:12:09	22.77	7.86	8.15
7/01/97	15:12:34	22.76	7.86	8.06
7/01/97	15:12:58	22.92	7.78	7.98

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Date mm/dd/yy	Time hh:mm:ss	Temperature C	DO mg/L	pH
7/02/97	9:51:41	22.33	8.48	8.13
7/02/97	9:52:17	22.38	8.24	8.11
7/02/97	9:54:57	22.61	7.72	8.15
7/02/97	9:55:28	22.53	7.79	8.06
7/02/97	9:55:59	22.62	7.79	8.15
7/02/97	9:57:09	22.60	7.32	8.12
7/02/97	9:57:37	22.64	7.46	8.10
7/02/97	9:58:08	22.62	7.63	8.29
7/02/97	9:58:30	22.71	7.68	7.99
7/02/97	9:59:35	22.65	7.35	8.30
7/02/97	9:59:57	22.66	7.52	8.38 -
7/02/97	10:00:28	22.61	7.80	8.23
7/02/97	10:00:52	22.59	7.95	8.06
7/02/97	10:01:47	22.53	8.11	8.07
7/02/97	10:02:02	22.59	8.08	8.10
7/02/97	10:02:22	22.55	8.10	8.09
7/02/97	10:02:52	22.62	8.07	8.25
7/02/97	10:03:57	22.66	7.50	7.97
7/02/97	10:04:27	22.67	7.51	7.93
7/02/97	10:04:54	22.65	7.53	8.12
7/02/97	10:05:19	22.65	7.72	8.29
7/02/97	10:06:06	22.54	7.68	8.01
7/02/97	10:06:34	22.64	7.65	8.03
7/02/97	10:07:09	22.70	7.74	7.97
7/02/97	10:07:41	22.74	7.77	8.02
7/02/97	10:08:34	22.62	7.57	8.21
7/02/97	10:08:51	22.71	7.56	8.26
7/02/97	10:09:18	22.69	7.67	8.08
7/02/97	10:11:04	22.69	7.69	8.02
7/02/97	10:11:37	22.78	7.77	8.30
7/02/97	10:12:08	22.72	7.87	8.12
7/02/97	10:12:38	22.72	7.93	8.17
7/02/97	10:13:48	22.68	8.11	7.94
7/02/97	10:14:16	22.73	7.98	7.97
7/02/97	10:14:42	22.61	7.91	8.01
7/02/97	10:15:12	22.69	7.85	8.05
7/02/97	10:16:17	22.64	8.08	8.03
7/02/97	10:16:47	22.70	8.03	8.08
7/02/97	10:17:17	22.67	8.01	8.24
7/02/97	10:17:42	22.66	8.00	8.28
7/02/97	10:18:48	22.65	8.13	8.30
7/02/97	10:19:17	22.68	8.13	8.12
7/02/97	10:19:43	22.50	8.16	8.25
7/02/97	10:20:14	22.70	8.11	8.09
7/02/97	10:21:13	22.67	8.19	8.28
7/02/97	10:21:40	22.66	8.19	8.11
7/02/97	10:22:05	22.60	8.17	8.10
7/02/97	10:22:37	22.67	8.11	7.98

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Date mm/dd/yy	Time hh:mm:ss	Temperature C	DO mg/L	pH
7/03/97	10:22:39	22.50	8.19	7.90
7/03/97	10:23:14	22.64	8.02	8.05
7/03/97	10:23:43	22.65	7.89	8.18
7/03/97	10:24:07	22.63	7.86	8.04
7/03/97	10:24:31	22.67	7.80	8.15
7/03/97	10:25:26	22.69	7.71	8.13
7/03/97	10:25:47	22.70	7.72	8.05
7/03/97	10:26:06	22.66	7.72	8.30
7/03/97	10:26:34	22.76	7.71	8.08
7/03/97	10:27:32	22.73	7.58	8.33
7/03/97	10:27:57	22.69	7.67	8.48
7/03/97	10:28:25	22.63	7.90	8.25
7/03/97	10:28:47	22.61	8.00	8.10
7/03/97	10:29:36	22.74	7.78	8.01
7/03/97	10:29:56	22.70	7.73	8.10
7/03/97	10:30:16	22.66	7.76	8.07
7/03/97	10:30:29	22.62	7.78	8.21
7/03/97	10:31:29	22.72	7.64	7.93
7/03/97	10:31:47	22.72	7.53	7.86
7/03/97	10:32:09	22.66	7.45	8.09
7/03/97	10:32:35	22.66	7.57	8.40
7/03/97	10:33:23	22.67	7.69	8.03
7/03/97	10:33:47	22.66	7.66	8.09
7/03/97	10:34:07	22.60	7.72	7.95
7/03/97	10:34:25	22.54	7.72	8.03
7/03/97	10:35:18	22.66	7.81	8.23
7/03/97	10:35:38	22.66	7.73	8.29
7/03/97	10:36:16	22.76	7.63	7.99
7/03/97	10:39:54	22.77	7.35	7.90
7/03/97	10:40:18	22.86	7.27	8.27
7/03/97	10:40:58	22.78	7.59	8.14
7/03/97	10:41:25	22.78	7.75	8.20
7/03/97	10:42:14	22.69	7.91	7.96
7/03/97	10:42:40	22.68	7.83	7.95
7/03/97	10:42:54	22.63	7.81	7.98
7/03/97	10:43:17	22.65	7.78	8.08
7/03/97	10:44:08	22.68	7.75	8.01
7/03/97	10:44:27	22.67	7.73	8.13
7/03/97	10:44:46	22.71	7.78	8.27
7/03/97	10:45:08	22.67	7.85	8.34
7/03/97	10:46:04	22.82	7.80	8.32
7/03/97	10:46:23	22.70	7.85	8.17
7/03/97	10:46:40	22.64	7.90	8.25
7/03/97	10:47:06	22.66	7.94	8.16
7/03/97	10:47:48	22.76	7.87	8.30
7/03/97	10:48:08	22.66	7.93	8.19
7/03/97	10:48:56	22.76	7.93	8.00
7/03/97	10:49:17	22.71	7.81	8.01

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Date mm/dd/yy	Time hh:mm:ss	Temp C	SpCond uS/cm	Salinity PPT	DO mg/L	pH
7/04/97	7:42:21	22.51	527.00	0.2	7.14	7.72
7/04/97	7:42:31	22.51	577.00	0.3	7.18	8.06
7/04/97	7:42:39	22.60	553.00	0.3	7.17	8.17
7/04/97	7:42:49	22.67	552.00	0.3	7.16	8.19
7/04/97	7:42:56	22.68	609.00	0.3	7.17	8.16
7/04/97	7:43:55	22.53	539.00	0.3	7.26	8.28
7/04/97	7:44:01	22.58	616.00	0.3	7.25	8.21
7/04/97	7:44:08	22.66	583.00	0.3	7.24	8.25
7/04/97	7:44:14	22.71	527.00	0.2	7.22	8.35
7/04/97	7:45:18	22.55	594.00	0.3	7.28	8.42
7/04/97	7:45:24	22.61	581.00	0.3	7.29	8.47
7/04/97	7:45:31	22.66	612.00	0.3	7.28	8.48
7/04/97	7:45:38	22.69	586.00	0.3	7.29	8.34
7/04/97	7:46:30	22.60	549.00	0.3	7.28	8.19
7/04/97	7:46:36	22.64	525.00	0.2	7.29	8.20
7/04/97	7:46:42	22.68	534.00	0.3	7.25	8.23
7/04/97	7:46:48	22.70	608.00	0.3	7.26	8.26
7/04/97	7:47:38	22.62	564.00	0.3	7.28	8.16
7/04/97	7:47:45	22.65	556.00	0.3	7.28	8.11
7/04/97	7:47:51	22.69	555.00	0.3	7.24	8.10
7/04/97	7:47:56	22.70	580.00	0.3	7.21	8.24
7/04/97	7:48:45	22.59	568.00	0.3	7.23	8.20
7/04/97	7:48:51	22.63	540.00	0.3	7.21	8.18
7/04/97	7:48:57	22.68	528.00	0.3	7.19	8.16
7/04/97	7:49:03	22.69	539.00	0.3	7.18	8.13
7/04/97	7:51:19	22.51	587.00	0.3	7.28	8.32
7/04/97	7:51:25	22.59	585.00	0.3	7.25	8.31
7/04/97	7:51:32	22.68	609.00	0.3	7.20	8.24
7/04/97	7:53:29	22.54	605.00	0.3	7.21	7.47
7/04/97	7:53:37	22.67	599.00	0.3	7.14	7.88
7/04/97	7:54:13	22.72	532.00	0.3	7.09	8.19
7/04/97	7:54:20	22.77	544.00	0.3	7.11	8.20
7/04/97	7:55:06	22.68	567.00d	0.3	7.23	8.10
7/04/97	7:55:12	22.71	538.00	0.3	7.23	8.08
7/04/97	7:55:18	22.74	532.00	0.3	7.21	8.08
7/04/97	7:55:23	22.76	557.00	0.3	7.20	8.10
7/04/97	7:56:16	22.68	541.00	0.3	7.25	8.12
7/04/97	7:56:23	22.74	565.00	0.3	7.25	8.14
7/04/97	7:56:30	22.78	580.00	0.3	7.24	8.22
7/04/97	7:56:36	22.79	602.00	0.3	7.24	8.33
7/04/97	7:57:21	22.66	591.00	0.3	7.32	8.42
7/04/97	7:57:28	22.74	537.00	0.3	7.33	8.37
7/04/97	7:57:35	22.76	645.00	0.3	7.33	8.33
7/04/97	7:57:41	22.75	558.00	0.3	7.34	8.34
7/04/97	7:58:32	22.73	570.00	0.3	7.37	8.43
7/04/97	7:58:37	22.77	540.00	0.3	7.37	8.41
7/04/97	7:58:43	22.78	544.00	0.3	7.38	8.29
7/04/97	7:58:48	22.81	559.00	0.3	7.37	8.16

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Date mm/dd/yy	Time hh:mm:ss	Temp C	SpCond uS/cm	Salinity PPT	DO mg/L	pH
7/05/97	12:00:47	22.39	531.00	0.3	7.48	8.18
7/05/97	12:01:01	22.29	586.00	0.3	7.52	8.24
7/05/97	12:01:13	22.23	553.00	0.3	7.53	8.28
7/05/97	12:04:48	22.07	558.00	0.3	7.50	8.45
7/05/97	12:04:58	22.16	619.00	0.3	7.46	8.35
7/05/97	12:05:14	22.09	541.00	0.3	7.47	8.49
7/05/97	12:05:28	22.30	625.00	0.3	7.44	8.45
7/05/97	12:05:41	22.36	589.00	0.3	7.47	8.58
7/05/97	12:05:53	22.52	523.00	0.2	7.52	8.72
7/05/97	12:06:03	22.52	605.00	0.3	7.56	8.63
7/05/97	12:06:13	22.46	588.00	0.3	7.60	8.61
7/05/97	12:06:25	22.24	623.00	0.3	7.62	8.74
7/05/97	12:06:35	22.35	581.00	0.3	7.61	8.68
7/05/97	12:06:46	22.36	553.00	0.3	7.65	8.57
7/05/97	12:06:58	22.34	517.00	0.2	7.70	8.51
7/05/97	12:07:08	22.34	533.00	0.3	7.72	8.68
7/05/97	12:07:19	22.35	610.00	0.3	7.75	8.55
7/05/97	12:07:32	22.23	567.00	0.3	7.78	8.60
7/05/97	12:07:43	22.36	542.00	0.3	7.75	8.47
7/05/97	12:07:50	22.49	554.00	0.3	7.71	8.27
7/05/97	12:07:59	22.47	593.00	0.3	7.70	8.41
7/05/97	12:08:10	22.31	570.00	0.3	7.72	8.67
7/05/97	12:08:20	22.39	540.00	0.3	7.70	8.59
7/05/97	12:08:30	22.38	530.00	0.3	7.69	8.58
7/05/97	12:08:39	22.41	544.00	0.3	7.69	8.49
7/05/97	12:08:48	22.35	597.00	0.3	7.70	8.37
7/05/97	12:08:56	22.35	594.00	0.3	7.69	8.44
7/05/97	12:09:06	22.29	625.00	0.3	7.68	8.52
7/05/97	12:09:42	22.39	647.00	0.3	7.60	8.39
7/05/97	12:09:59	22.41	614.00	0.3	7.50	8.59
7/05/97	12:10:09	22.45	544.00	0.3	7.47	8.56
7/05/97	12:10:18	22.56	546.00	0.3	7.44	8.61
7/05/97	12:10:27	22.58	571.00	0.3	7.44	8.46
7/05/97	12:10:36	22.55	534.00	0.3	7.46	8.31
7/05/97	12:10:47	22.54	532.00	0.3	7.46	8.25
7/05/97	12:10:59	22.55	553.00	0.3	7.46	8.35
7/05/97	12:11:10	22.46	536.00	0.3	7.51	8.43
7/05/97	12:11:27	22.38	563.00	0.3	7.54	8.62
7/05/97	12:11:36	22.46	592.00	0.3	7.53	8.58
7/05/97	12:11:45	22.55	618.00	0.3	7.53	8.62
7/05/97	12:11:54	22.51	616.00	0.3	7.56	8.89
7/05/97	12:12:06	22.46	539.00	0.3	7.59	8.63
7/05/97	12:12:15	22.41	647.00	0.3	7.61	8.70
7/05/97	12:12:34	22.26	564.00	0.3	7.63	8.80
7/05/97	12:12:43	22.39	589.00	0.3	7.61	8.75
7/05/97	12:12:53	22.49	539.00	0.3	7.59	8.74
7/05/97	12:13:01	22.50	543.00	0.3	7.60	8.63
7/05/97	12:13:08	22.60	556.00	0.3	7.60	8.51

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Date mm/dd/yy	Time hh:mm:ss	Temp C	SpCond uS/cm	Salinity PPT	DO mg/L	pH
7/06/97	11:50:28	22.32	537.00	0.3	7.98	8.32
7/06/97	11:50:41	22.35	598.00	0.3	7.90	8.37
7/06/97	11:50:53	22.22	555.00	0.3	7.89	8.31
7/06/97	11:51:08	22.19	553.00	0.3	7.88	8.38
7/06/97	11:51:20	22.37	631.00	0.3	7.84	8.26
7/06/97	11:51:37	22.14	538.00	0.3	7.79	8.62
7/06/97	11:51:49	22.33	622.00	0.3	7.71	8.39
7/06/97	11:52:03	22.41	595.00	0.3	7.73	8.91
7/06/97	11:52:14	22.42	524.00	0.2	7.78	8.82
7/06/97	11:52:24	22.42	605.00	0.3	7.82	8.71
7/06/97	11:52:32	22.46	596.00	0.3	7.86	8.62
7/06/97	11:52:42	22.48	614.00	0.3	7.86	8.89
7/06/97	11:52:53	22.52	564.00	0.3	7.88	8.73
7/06/97	11:53:05	22.32	548.00	0.3	7.99	8.70
7/06/97	11:53:16	22.43	513.00	0.2	8.01	8.62
7/06/97	11:53:26	22.45	526.00	0.2	8.05	8.86
7/06/97	11:53:35	22.42	611.00	0.3	8.08	8.61
7/06/97	11:53:45	22.42	560.00	0.3	8.10	8.72
7/06/97	11:53:55	22.42	549.00	0.3	8.11	8.51
7/06/97	11:54:06	22.43	550.00	0.3	8.09	8.38
7/06/97	11:54:18	22.30	590.00	0.3	8.11	8.58
7/06/97	11:54:29	22.24	570.00	0.3	8.11	8.78
7/06/97	11:54:37	22.35	534.00	0.3	8.08	8.59
7/06/97	11:54:47	22.20	529.00	0.3	8.11	8.57
7/06/97	11:54:58	22.34	541.00	0.3	8.07	8.45
7/06/97	11:55:08	22.36	597.00	0.3	8.05	8.50
7/06/97	11:55:17	22.49	600.00	0.3	8.01	8.58
7/06/97	11:55:26	22.49	628.00	0.3	8.00	8.57
7/06/97	11:56:10	20.16 ⓐ	689.00	0.3	8.40	8.45
7/06/97	11:56:25	22.23	617.00	0.3	7.84	8.37
7/06/97	11:56:36	22.46	542.00	0.3	7.75	8.52
7/06/97	11:56:48	22.62	549.00	0.3	7.71	8.47
7/06/97	11:56:59	22.60	571.00	0.3	7.73	8.41
7/06/97	11:57:09	22.58	531.00	0.3	7.74	8.32
7/06/97	11:57:22	22.48	530.00	0.3	7.77	8.51
7/06/97	11:57:33	22.59	551.00	0.3	7.77	8.47
7/06/97	11:57:43	22.59	532.00	0.3	7.79	8.46
7/06/97	11:58:03	22.44	557.00	0.3	7.84	8.75
7/06/97	11:58:18	22.30	596.00	0.3	7.90	8.89
7/06/97	11:58:31	22.48	627.00	0.3	7.89	8.79
7/06/97	11:58:41	22.44	626.00	0.3	7.91	8.84
7/06/97	11:58:56	22.39	540.00	0.3	7.90	8.61
7/06/97	11:59:07	22.49	686.00	0.3	7.87	8.57
7/06/97	11:59:17	22.51	563.00	0.3	7.86	8.59
7/06/97	11:59:29	22.51	588.00	0.3	7.86	8.67
7/06/97	11:59:42	22.62	532.00	0.3	7.86	8.80
7/06/97	11:59:54	22.42	538.00	0.3	7.93	8.81
7/06/97	12:00:04	22.59	552.00	0.3	7.92	8.66

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22.37 tan

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Date mm/dd/yy	Time hh:mm:ss	Temp C	SpCond uS/cm	Salinity PPT	DO mg/L	pH
7/07/97	13:18:55	22.42	540.00	0.3	6.58	8.00
7/07/97	13:19:35	22.48	605.00	0.3	7.41	8.38
7/07/97	13:19:57	23.02	506.00	0.2	7.26	8.25
7/07/97	13:20:37	22.67	544.00	0.3	7.52	8.31
7/07/97	13:20:55	23.10	574.00	0.3	7.40	8.28
7/07/97	13:21:36	22.73	525.00	0.2	7.67	8.56
7/07/97	13:22:00	23.26	572.00	0.3	7.61	8.27
7/07/97	13:23:54	23.02	552.00	0.3	7.80	8.37
7/07/97	13:24:12	23.19	488.00	0.2	7.86	8.42
7/07/97	13:24:50	22.79	608.00	0.3	7.98	8.66
7/07/97	13:25:15	22.84	588.00	0.3	7.74	9.06
7/07/97	13:26:06	23.02	577.00	0.3	7.89	8.12
7/07/97	13:26:29	23.05	515.00	0.2	7.39	8.51
7/07/97	13:27:06	22.87	511.00	0.2	7.48	8.21
7/07/97	13:27:16	22.87	506.00	0.2	7.69	8.59
7/07/97	13:28:15	22.66	525.00	0.2	7.93	8.83
7/07/97	13:28:31	22.93	550.00	0.3	7.92	8.58
7/07/97	13:29:00	22.74	553.00	0.3	8.02	8.69
7/07/97	13:29:18	23.03	513.00	0.2	8.01	8.39
7/07/97	13:30:04	22.81	534.00	0.3	7.99	8.04
7/07/97	13:30:15	22.81	585.00	0.3	7.96	8.79
7/07/97	13:30:47	22.84	541.00	0.3	7.95	8.33
7/07/97	13:30:55	22.86	527.00	0.2	7.16	8.49
7/07/97	13:31:24	22.74	523.00	0.2	7.80	8.59
7/07/97	13:31:35	22.81	539.00	0.3	7.83	8.62
7/07/97	13:32:10	22.92	576.00	0.3	7.87	8.40
7/07/97	13:32:22	23.05	592.00	0.3	7.81	8.72
7/07/97	13:32:33	23.15	629.00	0.3	7.81	8.72
7/07/97	13:33:32	22.65	635.00	0.3	7.75	7.71
7/07/97	13:33:43	22.96	616.00	0.3	7.13	8.58
7/07/97	13:34:12	22.90	541.00	0.3	7.67	8.67
7/07/97	13:34:23	23.00	548.00	0.3	7.67	8.63
7/07/97	13:34:53	22.96	543.00	0.3	7.70	8.24
7/07/97	13:35:02	23.04	512.00	0.2	7.70	8.31
7/07/97	13:35:42	22.89	527.00	0.2	7.79	8.59
7/07/97	13:35:52	22.98	540.00	0.3	7.76	8.70
7/07/97	13:36:35	22.96	499.00	0.2	7.77	8.12
7/07/97	13:36:55	22.97	546.00	0.3	7.72	8.84
7/07/97	13:37:31	22.90	599.00	0.3	7.88	8.99
7/07/97	13:37:40	22.92	614.00	0.3	7.89	9.04
7/07/97	13:38:11	22.86	588.00	0.3	7.91	8.61
7/07/97	13:38:19	22.91	535.00	0.3	7.25	8.65
7/07/97	13:38:59	22.77	709.00	0.4	7.69	8.58
7/07/97	13:39:11	22.85	567.00	0.3	6.87	8.68
7/07/97	13:39:43	22.90	594.00	0.3	7.75	8.96
7/07/97	13:39:52	22.95	530.00	0.3	7.77	9.06
7/07/97	13:40:29	22.73	533.00	0.3	7.89	8.90
7/07/97	13:40:38	22.91	548.00	0.3	7.85	8.95

YSI 6000 Time Series Report

Date mm/dd/yy	Time hh:mm:ss	Temperature C	DO mg/L	pH
7/08/97	10:57:26	21.98	8.29	7.99
7/08/97	10:57:55	22.09	8.11	8.20
7/08/97	10:58:20	22.24	8.02	8.24
7/08/97	10:59:01	22.25	8.00	8.10
7/08/97	10:59:20	22.13	7.98	8.24
7/08/97	11:00:44	22.23	8.09	8.17
7/08/97	11:01:34	22.35	8.25	8.35
7/08/97	11:02:12	22.41	8.35	8.69
7/08/97	11:02:44	22.50	8.33	8.38
7/08/97	11:04:01	22.33	8.21	8.39
7/08/97	11:04:33	22.40	8.15	8.60
7/08/97	11:04:59	22.34	8.21	8.40
7/08/97	11:05:18	22.42	8.26	8.49
7/08/97	11:06:19	22.33	8.34	8.23
7/08/97	11:06:49	22.38	8.28	8.41
7/08/97	11:07:13	22.36	8.31	8.41
7/08/97	11:07:39	22.36	8.32	8.36
7/08/97	11:08:39	22.36	8.26	8.26
7/08/97	11:08:59	22.37	8.33	8.23
7/08/97	11:09:24	22.42	8.41	8.22
7/08/97	11:09:46	22.36	8.41	8.48
7/08/97	11:10:47	22.46	8.16	8.24
7/08/97	11:11:16	22.47	8.16	8.28
7/08/97	11:11:55	22.53	8.21	8.16
7/08/97	11:12:09	22.37	8.24	8.16
7/08/97	11:12:53	22.62	8.11	8.27
7/08/97	11:13:20	22.76	8.03	8.43
7/08/97	11:13:53	22.68	8.07	8.23
7/08/97	11:15:00	22.48	8.04	8.19
7/08/97	11:15:18	22.47	8.07	8.34
7/08/97	11:15:57	22.48	8.08	8.23
7/08/97	11:16:22	22.46	8.10	8.22
7/08/97	11:17:10	22.37	8.09	8.08
7/08/97	11:17:33	22.36	8.07	8.08
7/08/97	11:18:00	22.49	8.03	8.24
7/08/97	11:18:23	22.47	8.07	8.30
7/08/97	11:19:22	22.47	8.09	8.06
7/08/97	11:19:43	22.43	8.06	8.43
7/08/97	11:20:12	22.53	8.12	8.52
7/08/97	11:20:37	22.47	8.18	8.43
7/08/97	11:22:01	22.54	8.08	8.37
7/08/97	11:22:33	22.53	8.07	8.23
7/08/97	11:23:03	22.45	8.09	8.30
7/08/97	11:23:19	22.37	8.08	8.24
7/08/97	11:24:21	22.49	8.05	8.49
7/08/97	11:24:43	22.46	8.11	8.51
7/08/97	11:25:02	22.50	8.17	8.39
7/08/97	11:25:26	22.60	8.27	8.48

YSI 6000 Time Series Report   Page 1

Date mm/dd/yy	Time hh:mm:ss	Temp C	SpCond uS/cm	Salinity PPT	DO mg/L	pH
7/09/97	7:40:08	22.02	554.00	0.3	7.66	7.96
7/09/97	7:40:16	22.11	624.00	0.3	7.57	8.22
7/09/97	7:40:23	22.21	569.00	0.3	7.52	8.37
7/09/97	7:40:33	22.31	551.00	0.3	7.47	8.38
7/09/97	7:40:39	22.33	616.00	0.3	7.43	8.35
7/09/97	7:40:46	22.35	533.00	0.3	7.40	8.46
7/09/97	7:40:52	22.37	578.00	0.3	7.39	8.47
7/09/97	7:41:01	22.40	587.00	0.3	7.37	8.64
7/09/97	7:41:07	22.45	538.00	0.3	7.37	8.74
7/09/97	7:41:13	22.49	614.00	0.3	7.37	8.68
7/09/97	7:41:19	22.51	596.00	0.3	7.37	8.75
7/09/97	7:41:25	22.51	594.00	0.3	7.38	8.74
7/09/97	7:41:31	22.49	541.00	0.3	7.39	8.69
7/09/97	7:41:37	22.48	558.00	0.3	7.41	8.63
7/09/97	7:41:42	22.49	522.00	0.2	7.41	8.59
7/09/97	7:41:47	22.50	540.00	0.3	7.42	8.65
7/09/97	7:41:52	22.51	584.00	0.3	7.42	8.65
7/09/97	7:41:59	22.51	564.00	0.3	7.43	8.59
7/09/97	7:42:04	22.52	546.00	0.3	7.44	8.53
7/09/97	7:42:09	22.52	563.00	0.3	7.45	8.45
7/09/97	7:42:15	22.54	590.00	0.3	7.44	8.51
7/09/97	7:42:21	22.54	583.00	0.3	7.43	8.64
7/09/97	7:42:27	22.56	548.00	0.3	7.43	8.60
7/09/97	7:42:34	22.56	540.00	0.3	7.43	8.52
7/09/97	7:42:39	22.57	554.00	0.3	7.43	8.45
7/09/97	7:42:45	22.56	597.00	0.3	7.43	8.49
7/09/97	7:42:50	22.59	603.00	0.3	7.41	8.56
7/09/97	7:42:55	22.63	622.00	0.3	7.38	8.59
7/09/97	7:43:28	22.29	653.00	0.3	7.44	8.54
7/09/97	7:43:33	22.40	629.00	0.3	7.42	8.58
7/09/97	7:43:40	22.47	550.00	0.3	7.40	8.66
7/09/97	7:43:45	22.52	550.00	0.3	7.39	8.64
7/09/97	7:43:51	22.53	568.00	0.3	7.41	8.50
7/09/97	7:43:57	22.53	523.00	0.2	7.42	8.43
7/09/97	7:44:04	22.52	542.00	0.3	7.42	8.48
7/09/97	7:44:10	22.54	533.00	0.3	7.40	8.52
7/09/97	7:44:18	22.54	518.00	0.2	7.41	8.48
7/09/97	7:44:24	22.54	557.00	0.3	7.41	8.51
7/09/97	7:44:32	22.54	612.00	0.3	7.41	8.66
7/09/97	7:44:38	22.58	627.00	0.3	7.41	8.74
7/09/97	7:44:45	22.58	621.00	0.3	7.43	8.76
7/09/97	7:44:51	22.59	548.00	0.3	7.45	8.72
7/09/97	7:44:57	22.59	686.00	0.3	7.46	8.67
7/09/97	7:45:07	22.57	581.00	0.3	7.48	8.65
7/09/97	7:45:15	22.57	603.00	0.3	7.48	8.65
7/09/97	7:45:22	22.60	548.00	0.3	7.48	8.77
7/09/97	7:45:28	22.58	542.00	0.3	7.49	8.81
7/09/97	7:45:38	22.60	554.00	0.3	7.49	8.77

AQUA SURVEY, INC.

CULTURE LAB DISTRIBUTION FORM

DATE: _6-25-97_

TEST JOB#: _97-296_   CLIENT: _Weston-REAL_

TEST LOCATION: IN-LAB [✓]   FIELD [   ]

TEST SPECIES: _H. azteca_

TOTAL NUMBER ORGANISMS TRANSFERRED: _1100+_

AQUA SURVEY, INC. CULTURE LAB INVESTIGATORS: _EL_

A. ORGANISMS

    1.   ASI CULTURE/HOLDING UNIT: _H. system_

    2.   RECEIVING LOG #: _N/A_

    3.   CULTURE LOG #: _97-0467_

    4.   AGE/SIZE INFORMATION: _2-3mm_

B. HOLDING [   ] CULTURE [✓] WATER PARAMETERS

    1.   TEMPERATURE: _22.0°C_

    2.   SALINITY: _N/A_

    3.   WATER SOURCE: _Well water_

C. TRANSFER CUSTODY & TRANSFER

    1.   LIVESTOCK RELINQUISHMENT DATE: _6-25-97_
                                        TIME: _1430hr_
                                        BY: _a_

    2.   LIVESTOCK RECEIVING   DATE: _6-25-97_
                                 TIME: _1430h.._
                                 BY: _SM_

    3. CULTURE SUPERVISOR OR SENIOR TECH. INITIALS: _a_

REMARKS: _____

_____

_____

Control Chart of Point Estimate Values
Acute SRT Tests with H. azteca

Figure axes: ug Cadmium (y-axis, 0 to 50) vs Test Number (April 96- June 97) (x-axis, 1 to 5)
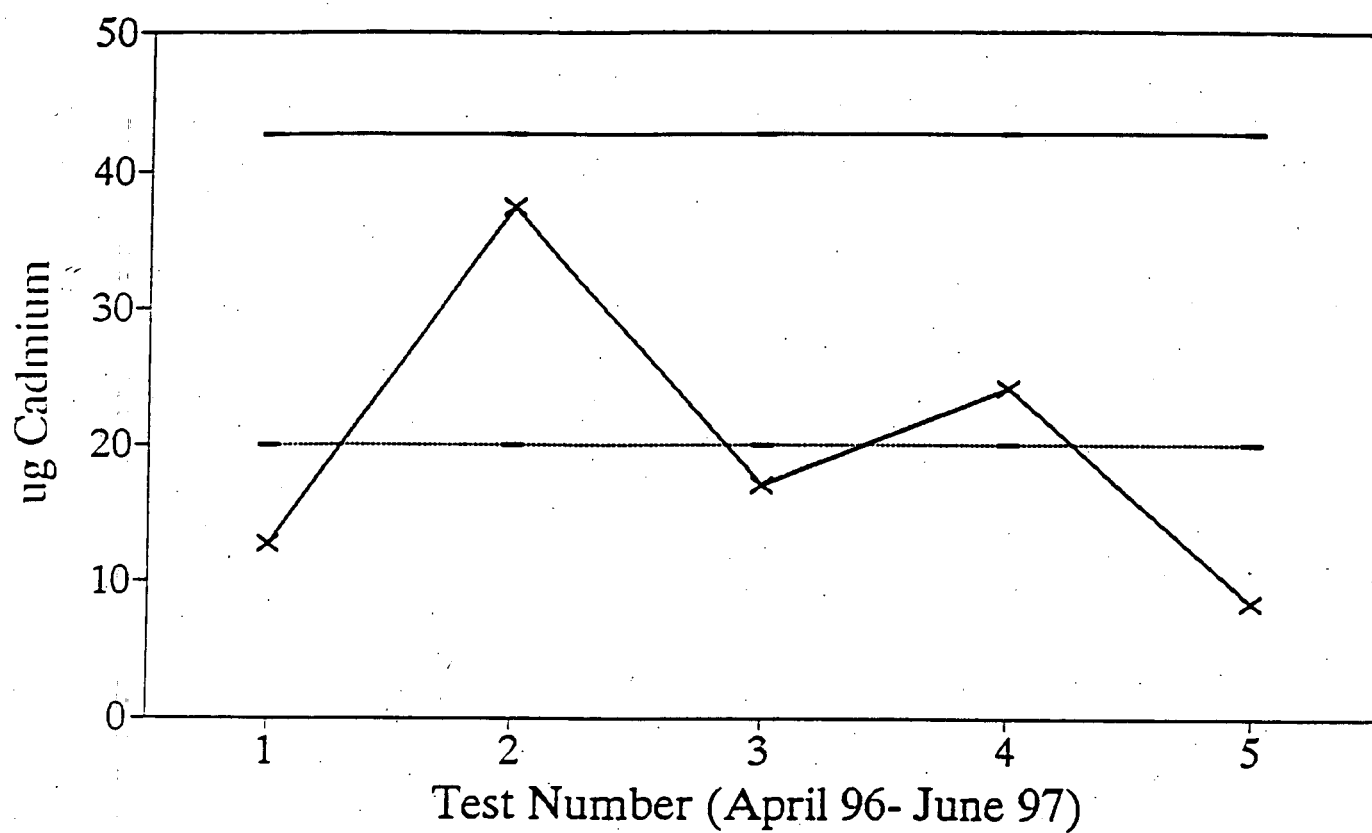
Legend: —×— LC50   ----- MEAN   —— Upper 95%   —— Lower 95%

Standard Reference Toxicant
Live Counts

For Job #: __97-286__   Start Date: __6/25/97__   End Date: _____

Organism __C. tentans/H. azteca__   Organism Log #: _____   Starting Time: __5:00p.__

Dose C	Initial	1	2	3	4	Dose	Initial	1	2	3	4
A	i	1	1	1	1	A 16.0	1	1	1	O	O
B	1	O	—	—	O	B	1	1	1	1	O
C	1	1	1	1	1	C	1	1	1	1	O
D	1	1	1	1	1	D	1	1	O	—	O
E	i	1	1	1	1	E	1	1	1	1	1
F	1	1	O	—	O	F	1	1	1	1	1
G	1	1	1	1	1	G	1	1	O	—	O
H	i	1	1	i	1	H	1	1	1	O	O
I	1	1	1	1	1	I	.	1	1	1	O
J	1	1	1	i	1	J	.	1	1	O	O
A 8.0	1	1	1	1	1	A 20.0	1	1	O	—	O
B	1	1	1	1	i	B	i	1	O	—	O
C	1	1	1	1	1	C	1	i	O	—	O
D	1	1	1	O	O	D	1	1	O	—	O
E	1	1	1	1	O	E	1	1	O	—	O
F	1	1	1	i	1	F	1	1	O	—	O
G	1	1	1	i	1	G	1	1	O	—	O
H	1	1	1	1	1	H	1	1	O	—	O
I	1	1	1	1	1	I	1	O	—	—	O
J	1	1	1	1	1	J	1	O	—	—	
A 0.0	1	O	O	—	O	A 64.0	1	O	—	—	O
B	1	1	1	.	1	B	1	O	—	—	O
C	1	1	1	1	1	C	1	O	—	—	O
D	1	O	O	—	O	D	1	O	—	—	O
E	1	i	1	1	1	E	1	O	—	—	O
F	1	O	O	—	O	F	1	1	O	—	O
G	1	1	1	1	1	G	1	1	O	—	O
H	1	1	1	1	1	H	1	O	—	—	O
I	1	1	1	1	1	I	1	O	—	—	O
J	1	1	O	—	O	J	1	O	—	—	O

Initial	TO	JH	JH	JH	JH		TO	JH	JH	JH	JH
Date	6/25	6/26	6/27	6/28	6/29		6/25	6/26	6/27	6/28	6/29

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SAMPLE ID: SRT H. azteca 6/25/97 97-286

CONCENTRATION	NUMBER EXPOSED	NUMBER AFFECTED	PERCENT AFFECTED	BINOMIAL PROBABILITY (%)
64.0000	10	10	100.0000000	0.0976563
32.0000	10	10	100.0000000	0.0976563
16.0000	10	8	80.0000000	5.4687500
8.0000	10	4	40.0000000	37.6953100
4.0000	10	2	20.0000000	5.4687500

-------------------------------- RESULTS --------------------------------------

METHOD	SPAN	G	EC50	95 PERCENT CONFIDENCE LIMITS	
BINOMIAL			9.449324	0.000000 -	32.000000
MOVING AVERAGE	3	0.214882	8.571264	5.130138 -	12.301770
PROBIT		0.252222	8.331825	5.400714 -	11.864480

─────────── INTERPRETATION OF STATISTICS ───────────

The order of preference of results is:
    BEST: Probit Method
    BETTER: Moving Average
    GOOD: Binomial
    GOOD: Graphic Interpolation (Graph drawn by hand)
assuming all are statistically valid.

To Determine if a method has given valid results:
GRAPHICAL: Always valid
BINOMIAL: The program will tell you if this is not valid.
MOVING AVERAGE: The number of spans needs to be no less than one
                 lower than the number of concentrations tested
                 (excluding control).
PROBIT: The program will tell you if this is not valid

YSI 6000 Time Series Report

Date mm/dd/yy	Time hh:mm:ss	Temp C	SpCond uS/cm	Salinity PPT	DO mg/L	pH
6/25/97	15:01:17 *0*	23.19	532.00	0.3	8.11	8.06
6/25/97	15:01:30 *4.0*	23.20	532.00	0.3	7.81	8.11
6/25/97	15:01:43 *8.0*	23.16	524.00	0.2	7.79	8.14
6/25/97	15:01:54 *16.0*	23.16	514.00	0.2	7.77	8.14
6/25/97	15:02:07 *32.0*	23.21	493.00	0.2	7.74	8.14
6/25/97	15:02:20 *64.0*	23.41	430.00	0.2	7.68	8.16

Project No. _97-286_ , Day of Test, _0hr._ , Date _6/25/97_

Species _H. azteca_   Test Type _SRT_

Acceptable Range ✓ if OK

Salinity: _N/A_ To _____ ✓

Temperature: _22_ To _24_ ✓

pH: _7.3_ To _8.3_ ✓

Dissolved Oxygen: _> 5.0_ ✓

Actions Taken: _____

See Deviation Summary. Tech. Init. _AW_

YSI 6000 Time Series Report   Page 1

Date mm/dd/yy	Time hh:mm:ss	Temp C	SpCond uS/cm	Salinity PPT	DO mg/L	pH
6/26/97	13:18:51 *0*	22.59	564.00	0.3	7.70	8.11
6/26/97	13:19:04 *4.0*	22.57	558.00	0.3	7.52	8.08
6/26/97	13:19:13 *8.0*	22.55	552.00	0.3	7.44	8.08
6/26/97	13:19:23 *16.0*	22.51	543.00	0.3	6.76	8.07
6/26/97	13:19:31 *32.0*	22.51	520.00	0.2	6.73	8.06
6/26/97	13:19:39 *64.0*	22.50	455.00	0.2	6.70	8.04

97-286   *24hr*   *6/26/97*

Series *H. azteca*   Test Type *SRT*

Acceptable Range

_____ *N/A* To _____ ✓

Temperature: *22* To *24* ✓

7.3 To *8.3* ✓

Dissolved Oxygen: > *5.0* ✓

Time Taken: *none*

CAN

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Date mm/dd/yy	Time hh:mm:ss	Temperature C	DO mg/L	pH
6/27/97	10:27:14	22.60	7.63	8.21
6/27/97	10:27:35	22.66	7.53	8.21
6/27/97	10:27:52	22.64	7.50	8.21
6/27/97	10:28:15	22.59	7.51	8.20
6/27/97	10:28:28	22.57	7.51	8.18
6/27/97	10:28:42	22.56	7.52	8.16

Tank No. _286_   Day of Study ___48 hc__ , Date _6/27/_

Species ___Ha___   Test Type ___SRT___

Acceptable Range, ✓ if o

Salinity:	_____ To _____	
Temperature:	__22_ To _24_	✓
pH:	_7.3_ To _8.3_	✓
Dissolved Oxygen:	_____ > _5_	✓
Actions Taken:		

Technician Initials _MW_

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YSI 6000 Time Series Report   Page 1

Date mm/dd/yy	Time hh:mm:ss	Temperature C	DO mg/L	pH
6/28/97	10:40:12	22.53	7.55	8.24
6/28/97	10:40:35	22.70	7.34	8.25
6/28/97	10:40:53	22.70	7.29	8.25
6/28/97	10:41:03	22.68	7.27	8.23

Project No. 97-286   Duration of Study ___72hr___ , Date 6/29

Species ___Ha.___ Test Type ___SRT___

Acceptable Range ✓ if OK

Salinity	___	To ___	___
Temperature:	___ 22	Tc .24/	✓
pH:	___ 7.3	Tc 8.3	✓
Dissolved Oxygen:	___	> 5	✓
Actions Taken:			

See Deviation Summary Sheet   Initials: ___JAL___

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YSI 6000 Time Series Report   Page 1

Date mm/dd/yy	Time hh:mm:ss	Temperature C	DO mg/L	pH
6/29/97	11:12:58	22.57	7.54	8.09
6/29/97	11:13:10	22.62	7.47	8.20
6/29/97	11:13:23	22.64	7.46	8.21
6/29/97	11:13:36	22.62	7.46	8.21

97-286   Dev. of State: 966-   Date 6/29

Diseahess   H.a.   Test Type SRT

Acceptable Range

Salinity:	___ To ___	___
Temperature:	22 To 24	✓
pH:	7.3 To 8.3	✓
Dissolved Oxygen:	> 5	✓
Actions Taken:		

AQUA SURVEY, INC.

CULTURE LAB DISTRIBUTION FORM

DATE: _6-25-97_

TEST JOB#: _SET_   CLIENT: _In house_

TEST LOCATION: IN-LAB [✓]   FIELD [   ]

TEST SPECIES: _H. azteca_

TOTAL NUMBER ORGANISMS TRANSFERRED: _10⁴_

AQUA SURVEY, INC. CULTURE LAB INVESTIGATORS: _EL_

A.   **ORGANISMS**

1.   ASI CULTURE/HOLDING UNIT: _H system_

2.   RECEIVING LOG #:   _N/A_

3.   CULTURE LOG #: _97-0468_

4.   AGE/SIZE INFORMATION: _2-3 mm_

B.   **HOLDING** [   ] **CULTURE** [✓] **WATER PARAMETERS**

1.   TEMPERATURE: _22.0°C_

2.   SALINITY: _N/A_

3.   WATER SOURCE: _Well water_

C.   **TRANSFER CUSTODY & TRANSFER**

1.   LIVESTOCK RELINQUISHMENT DATE: _6-25-97_
                                    TIME: _1130hr_
                                    BY: _EL_

2.   LIVESTOCK RECEIVING   DATE: _6-25-97_
                                    TIME: _1430hr_
                                    BY: _ST_

3.   CULTURE SUPERVISOR OR SENIOR TECH. INITIALS: _____

REMARKS: _____

_____

_____

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Recorder ID: 7003214   Deployment #: 31   Interval: 1 hour   Samples: 361
Start: 06/24/97 15:44:27   Recover: 07/09/97 16:52:05   Min/Max Window: 18/ 22C
Description: Water bath room H. azteca Job # 97-286

State: Deployed

Overall Summary

Data File: 7003214.031

    Temperature Extremes: 23.5 C   14:44:27   06/25/97
                            21.0 C   16:44:27   06/25/97

^ Over Temperature Window
  Time: 5 days 00:00:00
  # of Samples: 120

' Under Temperature Window
  Time: 0 days 00:00:00
  # of Samples: 0

Daily Summary

Date	# Samples	Min Temp C	Max Temp C	# Under Window	# Over Window
06/24/97	8	22.5	23.0	0	8
6/25/97	24	21.0	23.5	0	15
6/26/97	24	21.5	22.5	0	8
06/27/97	24	22.0	22.5	0	9
06/28/97	24	22.0	22.5	0	5
6/29/97	24	22.0	22.5	0	12
06/30/97	24	21.5	22.5	0	10
07/01/97	24	22.0	22.5	0	5
7/02/97	24	21.5	22.5	0	5
7/03/97	24	21.5	22.5	0	4
07/04/97	24	22.0	22.5	0	6
7/05/97	24	22.0	22.5	0	11
7/06/97	24	22.0	22.5	0	13
07/07/97	24	22.0	22.5	0	6
07/08/97	24	21.5	22.5	0	3
7/09/97	17	22.0	22.0	0	0

Instrument Data

Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C
6/24/97							
16:44:27 ^	22.5	17:44:27 ^	23.0	18:44:27 ^	23.0	19:44:27 ^	23.0
20:44:27 ^	23.0	21:44:27 ^	23.0	22:44:27 ^	23.0	23:44:27 ^	22.5
06/25/97							
00:44:27 ^	22.5	01:44:27 ^	22.5	02:44:27 ^	22.5	03:44:27 ^	22.5
4:44:27 ^	22.5	05:44:27 ^	22.5	06:44:27 ^	22.5	07:44:27 ^	22.5
8:44:27 ^	22.5	09:44:27 ^	22.5	10:44:27 ^	22.5	11:44:27 ^	23.0
12:44:27 ^	23.0	13:44:27 ^	23.0	14:44:27 ^	23.5	15:44:27	22.0
6:44:27	21.0	17:44:27	21.0	18:44:27	22.0	19:44:27	21.5
0:44:27	22.0	21:44:27	22.0	22:44:27	22.0	23:44:27	22.0
06/26/97							
0:44:27 ^	22.5	01:44:27	22.0	02:44:27	22.0	03:44:27 ^	22.5
04:44:27	22.0	05:44:27	22.0	06:44:27	22.0	07:44:27	22.0

Recorder ID: 7003214   Deployment #: 31   Interval: 1 hour   Samples: 361
Start: 06/24/97 15:44:27   Recover: 07/09/97 16:52:05   Min/Max Window: 18/ 22
Description: Water bath room H. azteca Job # 97-286

State: Deployed

Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C
08:44:27	22.0	09:44:27	22.0	10:44:27	22.0	11:44:27	22.0
12:44:27	22.0	13:44:27	21.5	14:44:27	22.0	15:44:27	21.5
16:44:27	22.0	17:44:27 ^	22.5	18:44:27 ^	22.5	19:44:27 ^	22.5
20:44:27 ^	22.5	21:44:27 ^	22.5	22:44:27 ^	22.5	23:44:27	22.0

06/27/97

Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C
00:44:27	22.0	01:44:27 ^	22.5	02:44:27 ^	22.5	03:44:27	22.0
04:44:27 ^	22.5	05:44:27 ^	22.5	06:44:27 ^	22.5	07:44:27 ^	22.5
08:44:27 ^	22.5	09:44:27 ^	22.5	10:44:27 ^	22.5	11:44:27	22.0
12:44:27	22.0	13:44:27	22.0	14:44:27	22.0	15:44:27	22.0
16:44:27	22.0	17:44:27	22.0	18:44:27	22.0	19:44:27	22.0
20:44:27	22.0	21:44:27	22.0	22:44:27	22.0	23:44:27	22.0

06/28/97

Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C
00:44:27	22.0	01:44:27	22.0	02:44:27	22.0	03:44:27	22.0
04:44:27	22.0	05:44:27	22.0	06:44:27	22.0	07:44:27	22.0
08:44:27	22.0	09:44:27	22.0	10:44:27	22.0	11:44:27	22.0
12:44:27	22.0	13:44:27 ^	22.5	14:44:27 ^	22.5	15:44:27	22.0
16:44:27	22.0	17:44:27	22.0	18:44:27	22.0	19:44:27	22.0
20:44:27 ^	22.5	21:44:27 ^	22.5	22:44:27	22.0	23:44:27 ^	22.5

06/29/97

Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C
00:44:27 ^	22.5	01:44:27 ^	22.5	02:44:27 ^	22.5	03:44:27 ^	22.5
04:44:27	22.0	05:44:27	22.0	06:44:27	22.0	07:44:27	22.0
08:44:27 ^	22.5	09:44:27	22.0	10:44:27	22.0	11:44:27 ^	22.5
12:44:27 ^	22.5	13:44:27	22.0	14:44:27	22.0	15:44:27	22.0
16:44:27	22.0	17:44:27	22.0	18:44:27 ^	22.5	19:44:27	22.0
20:44:27 ^	22.5	21:44:27 ^	22.5	22:44:27 ^	22.5	23:44:27 ^	22.5

06/30/97

Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C
00:44:27 ^	22.5	01:44:27 ^	22.5	02:44:27 ^	22.5	03:44:27 ^	22.5
04:44:27 ^	22.5	05:44:27	22.0	06:44:27 ^	22.5	07:44:27 ^	22.5
08:44:27	22.0	09:44:27 ^	22.5	10:44:27 ^	22.5	11:44:27	22.0
12:44:27	22.0	13:44:27 ^	22.5	14:44:27	22.0	15:44:27	22.0
16:44:27	22.0	17:44:27	22.0	18:44:27	22.0	19:44:27	22.0
20:44:27	21.5	21:44:27	22.0	22:44:27	21.5	23:44:27	22.0

07/01/97

Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C
00:44:27	22.0	01:44:27	22.0	02:44:27	22.0	03:44:27	22.0
04:44:27	22.0	05:44:27	22.0	06:44:27	22.0	07:44:27	22.0
08:44:27 ^	22.5	09:44:27 ^	22.5	10:44:27 ^	22.5	11:44:27 ^	22.5
12:44:27 ^	22.5	13:44:27	22.0	14:44:27	22.0	15:44:27	22.0
16:44:27	22.0	17:44:27	22.0	18:44:27	22.0	19:44:27	22.0
20:44:27	22.0	21:44:27	22.0	22:44:27	22.0	23:44:27	22.0

07/02/97

Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C
00:44:27	22.0	01:44:27	22.0	02:44:27	22.0	03:44:27	22.0
04:44:27	22.0	05:44:27	22.0	06:44:27	22.0	07:44:27	22.0
08:44:27	22.0	09:44:27	22.0	10:44:27 ^	22.5	11:44:27 ^	22.5
12:44:27 ^	22.5	13:44:27 ^	22.5	14:44:27	22.0	15:44:27	22.0
16:44:27	22.0	17:44:27 ^	22.5	18:44:27	22.0	19:44:27	22.0
20:44:27	21.5	21:44:27	22.0	22:44:27	21.5	23:44:27	22.0

Recorder ID: 7003214   Deployment #: 31   Interval: 1 hour   Samples: 361
Start: 06/24/97 15:44:27   Recover: 07/09/97 16:52:05   Min/Max Window: 18/ 22(

Description: Water bath room H. azteca Job # 97-286

State: Deployed

Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C
07/03/97							
00:44:27	22.0	01:44:27	21.5	02:44:27	22.0	03:44:27	21.5
04:44:27	22.0	05:44:27	22.0	06:44:27	22.0	07:44:27	22.0
08:44:27	22.0	09:44:27 ^	22.5	10:44:27	22.0	11:44:27 ^	22.5
12:44:27 ^	22.5	13:44:27 ^	22.5	14:44:27	22.0	15:44:27	22.0
16:44:27	22.0	17:44:27	22.0	18:44:27	22.0	19:44:27	22.0
20:44:27	22.0	21:44:27	21.5	22:44:27	22.0	23:44:27	21.5
07/04/97							
00:44:27	22.0	01:44:27	22.0	02:44:27	22.0	03:44:27	22.0
04:44:27	22.0	05:44:27	22.0	06:44:27	22.0	07:44:27	22.0
08:44:27 ^	22.5	09:44:27	22.0	10:44:27 ^	22.5	11:44:27	22.0
12:44:27	22.0	13:44:27 ^	22.5	14:44:27	22.0	15:44:27 ^	22.5
16:44:27	22.0	17:44:27 ^	22.5	18:44:27 ^	22.5	19:44:27	22.0
20:44:27	22.0	21:44:27	22.0	22:44:27	22.0	23:44:27	22.0
07/05/97							
00:44:27	22.0	01:44:27 ^	22.5	02:44:27	22.0	03:44:27 ^	22.5
04:44:27 ^	22.5	05:44:27 ^	22.5	06:44:27	22.0	07:44:27 ^	22.5
08:44:27	22.0	09:44:27	22.0	10:44:27	22.0	11:44:27 ^	22.5
12:44:27	22.0	13:44:27	22.0	14:44:27 ^	22.5	15:44:27	22.0
16:44:27 ^	22.5	17:44:27	22.0	18:44:27 ^	22.5	19:44:27	22.0
20:44:27	22.0	21:44:27	22.0	22:44:27 ^	22.5	23:44:27 ^	22.5
07/06/97							
00:44:27	22.0	01:44:27 ^	22.5	02:44:27 ^	22.5	03:44:27 ^	22.5
04:44:27 ^	22.5	05:44:27	22.0	06:44:27	22.0	07:44:27 ^	22.5
08:44:27 ^	22.5	09:44:27	22.0	10:44:27	22.0	11:44:27 ^	22.5
12:44:27	22.0	13:44:27 ^	22.5	14:44:27 ^	22.5	15:44:27 ^	22.5
16:44:27 ^	22.5	17:44:27	22.0	18:44:27 ^	22.5	19:44:27	22.0
20:44:27	22.0	21:44:27 ^	22.5	22:44:27	22.0	23:44:27	22.0
07/07/97							
00:44:27	22.0	01:44:27	22.0	02:44:27 ^	22.5	03:44:27	22.0
04:44:27	22.0	05:44:27	22.0	06:44:27	22.0	07:44:27 ^	22.5
08:44:27	22.0	09:44:27	22.0	10:44:27 ^	22.5	11:44:27 ^	22.5
12:44:27 ^	22.5	13:44:27	22.0	14:44:27	22.0	15:44:27	22.0
16:44:27	22.0	17:44:27	22.0	18:44:27 ^	22.5	19:44:27	22.0
20:44:27	22.0	21:44:27	22.0	22:44:27	22.0	23:44:27	22.0
07/08/97							
00:44:27	22.0	01:44:27	22.0	02:44:27	22.0	03:44:27	22.0
04:44:27 ^	22.5	05:44:27 ^	22.5	06:44:27	22.0	07:44:27 ^	22.5
08:44:27	22.0	09:44:27	21.5	10:44:27	21.5	11:44:27	21.5
12:44:27	22.0	13:44:27	22.0	14:44:27	22.0	15:44:27	22.0
16:44:27	22.0	17:44:27	22.0	18:44:27	22.0	19:44:27	22.0
20:44:27	22.0	21:44:27	22.0	22:44:27	22.0	23:44:27	22.0
7/09/97							
00:44:27	22.0	01:44:27	22.0	02:44:27	22.0	03:44:27	22.0
04:44:27	22.0	05:44:27	22.0	06:44:27	22.0	07:44:27	22.0
08:44:27	22.0	09:44:27	22.0	10:44:27	22.0	11:44:27	22.0

Recorder ID: 7003214   Deployment #: 31   Interval: 1 hour   Samples: 361
Start: 06/24/97 15:44:27   Recover: 07/09/97 16:52:05   Min/Max Window: 18/ 22
Description: Water bath room H. azteca Job # 97-286

State: Deployed

Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C	Date/Time	Temp C
12:44:27	22.0	13:44:27	22.0	14:44:27	22.0	15:44:27	22.0
16:44:27	22.0						

Chamber #

Pre-test	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
1	1.2	1.75 1.8	1.45	2.7	2.70	3.65	3.3	3.0	2.10	2.9	3.4	4.1	1.2	3.1	2.4	2.8	3.5	4.0	3.4	4.1	2.3	2.8	4.5	2.1	3.1	2.9
2	1.4	2.75	2.20	2.50	3.3	3.10	2.5	5.00	1.85	2.6	2.3	3.7	3.4	2.8	1.8	2.3	2.7	3.2	2.5	2.8	2.2	1.2	3.1	2.0	3.0	2.8
3	1.1	1.6	4.1	2.50	3.55	3.75	2.2	3.10	2.30	2.80	2.4	2.9	3.2	3.5	2.1	2.4	2.8	3.6	2.6	3.6	2.1	2.6	3.2	2.4	3.0	2.1
4	1.0	1.50	.85	3.50	3.10	3.55	2.2	2.9	2.0	2.40	2.6	3.2	3.6	2.8	2.2	2.8	3.2	4.1	3.1	3.5	2.5	2.5	2.6	2.3	1.8	2.9
5	1.3	1.95	3.95	2.0	3.55	3.1	2.4	5.20	2.8	2.90	2.5	2.6	3.3	3.5	2.4	2.2	2.2	3.3	3.4	3.3	2.6	2.1	3.1	2.6	2.2	2.7
6	1.6	2.80	3.05	1.90	2.10	1.7	2.85	3.10	2.70	1.90	2.7	3.1	4.2	2.6	2.1	2.4	2.3	3.1	3.2	2.9	2.2	2.8	2.1	3.1	2.2	2.2
7	1.9	1.65	2.55	3.40	2.50	3.6	3.15	3.55	5.00	2.30	1.9	2.4	2.9	3.5	2.3	2.7	2.6	2.9	3.2	3.2	3.1	2.9	2.2	3.2	2.1	1.8
8	1.2	.65	2.40	2.80	2.6	2.5	2.10	2.6	2.75	3.00	2.5	1.6	3.0	3.3	2.8	2.3	2.5	2.8	3.4	1.8	2.9	3.3	2.1	2.3	1.8	1.9
9	1.3	2.80	3.60	1.50	2.90	2.15	2.60	2.5	1.40	2.00	3.6	2.4	2.8	2.4	2.3	2.6	2.2	3.1	2.8	2.1	2.8	3.1	2.2	2.2	2.4	2.0
10	1.4	2.30	2.1	3.10	2.55	2.5	2.80	2.45	1.60	2.10	2.2	2.5	2.9	2.6	2.1	3.1	2.4	2.3	2.7	2.3	2.4	2.5	2.3	1.9	1.8	2.1
11	1.3	2.15	2.0	1.90	2.65	2.9	2.20	2.45	1.50	2.60	2.9	2.1	3.0	2.9	2.2	3.6	2.6	2.5	3.3	2.3	2.2	2.6	1.8	2.8	2.2	2.0
12	1.1	2.5	3.50	2.6	2.15	2.3	2.7	2.60	1.25	2.50	3.1	3.4	3.6	2.7	1.9	2.2	2.2	2.0	3.6	2.5	2.3		2.3	2.6	2.0	2.8
13	1.2	2.5	3.45	2.55	2.4	2.50	2.75	1.50	2.70	2.4	2.0	1.9	2.7	1.8	2.1	2.2	2.2	2.3	2.5	2.3			2.4	2.2	1.8	2.1
14	1.6	5.0	3.3	2.85	3.0	2.2	2.10	2.45	1.45	1.50	3.4	1.4	2.8	2.6	1.9	2.2	2.4	2.4		2.6	2.4		2.2	2.2	1.2	2.2
15	2.1	2.90	3.00	2.10	2.6	1.4	1.85	2.75	2.05	2.40	2.7	2.6	2.2	2.1	1.7	2.1	2.2	2.3		2.9		2.6	2.0	1.2	2.2	
16	1.4	2.0	1.90	1.6	3.25	2.35	2.10	2.00	2.70		3.8	3.3	3.2	2.1	1.8	2.3	2.2	2.9		2.6		2.9	2.6	1.7	1.6	
17	1.3	2.0	1.95	3.6	2.50	2.1	1.70	2.10	2.00		2.4	2.1	2.8	2.9	1.8	2.2	2.1	2.1		2.1		2.3	2.8	1.8	2.1	
18	1.1	2.30		2.65	3.0		1.95	1.85			1.5	2.9	2.7	1.9		2.6			2.6			2.4	2.2	2.4		
19	1.2	2.45			2.75						2.4	2.3	2.1		2.2			2.7					2.3			
20	1.6										2.2	2.0		2.5									2.2			

mm

| X̄ | 1.4 | 2.1 | 2.6 | 2.5 | 2.7 | 2.7 | 2.5 | 2.9 | 2.2 | 2.4 | 2.8 | 2.6 | 2.9 | 2.8 | 2.1 | 2.5 | 2.5 | 2.8 | 3.0 | 2.9 | 2.5 | 2.6 | 2.6 | 2.4 | 2.1 | 2.2 |

AN 7/27/97

date   2.8 QC   7/4 7/21 7/4
Initials   JN 11/11   I I I

① Chamber #9 only contained 15 organisms.   7/4
                                              I

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Is this a 5h → or it's a 3, the mean is not correct.

Chamber #

Pr-test	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
1	1.2	1.65	2.7	2.0	3.6	2.3	3.00	2.10	2.9	3.4	4.1	2.2	3.1	2.4	2.8	3.5	4.0	3.4	4.1	2.3	2.8	4.5	2.1	3.1	2.9
2	1.4	2.8 2.20	2.90	3.3 2.0	2.1	2.90	2.85	2.60	2.5	2.7	3.4	2.81	1.8	2.3	2.7	3.2	2.5	2.8	2.2	4.2	3.1	2.0	3.0	2.8
3	1.1	1.66	4.1	0.50	3.55	3.2	2.2	3.10	2.30	2.80	2.4	2.9	3.2	3.5	2.1	2.4	2.8	3.6	2.6	3.6	2.1	2.6	3.2	2.4	3.0	2.1
4	1.0	1.50	.55	3.5	3.10	3.55	2.0	2.93	2.0	3.40	2.6	2.3	3.6	2.8	2.2	2.0	3.2	4.1	3.1	3.5	2.5	2.5	2.6	2.3	4.8	2.9
5	1.3	1.95	3.95	7.0	3.55	3.1	2.4	5.00	2.8	2.90	2.5	2.6	3.3	3.5	2.4	2.9	2.2	3.3	3.4	3.3	2.6	2.1	3.1	2.6	2.2	2.7
6	1.6	2.0	2.05	1.90	2.40	1.9	2.85	3.40	2.90	1.90	2.7	3.4	4.2	2.6	2.1	2.4	2.3	3.1	3.2	2.9	2.2	2.8	2.1	3.1	2.2	2.2
7	1.9	1.65	2.55	3.40	2.50	3.45	3.45	3.55	3.00	2.10	1.4	2.4	7.9	3.5	2.3	2.7	7.6	2.9	3.2	3.1	3.1	2.2	3.2	2.1	1.8
8	1.2	2.6	2.90	8.2	2.60	2.5	2.10	2.65	2.75	3.00	2.5	1.6	3.0	3.3	2.8	2.3	2.5	2.8	3.4	1.8	2.9	3.3	2.1	2.3	1.8	1.9
9	1.3	2.8 3.60	1.50	2.90	2.15	2.60	3.05	1.90	2.00	3.6	2.4	2.8	2.4	2.3	2.6	2.2	3.1	2.8	2.1	2.8	3.1	2.2	2.2	2.4	2.0
10	1.4	2.3	2.1	3.10	2.60	2.5	2.9	2.95	1.60	1.10	2.2	2.5	2.1	2.6	2.1	3.1	2.4	2.3	2.7	2.3	2.4	2.5	2.3	1.7	1.8	2.1
11	1.3	2.05	2.01	1.7	2.65	2.3	2.65	1.90	2.60	2.9	2.1	3.0	2.9	2.2	3.6	2.6	2.5	3.3	2.3	2.2	2.6	1.8	2.6	2.2	2.0
12	1.1	2.5	3.30	3.60	2.15	2.3	2.70	2.60	1.75	1.50	3.1	3.4	3.6	2.7	1.9	2.2	2.2	2.0	3.6	2.5	2.3		2.3	2.6	2.0	1.8
13	1.2	2.5	3.45	2.2	2.35	2.4	2.50	2.95	1.50	2.90	2.4	2.2	1.9	2.7	1.8	2.1	2.2	2.2	2.3	2.5	2.3		2.4	2.2	1.8	2.1
14	1.6	2.0	2.3	2.35	3.0	2.2	2.10	2.45	1.45	1.50	3.4	1.4	2.8	2.6	1.9	2.2	2.4	2.4		2.6	2.4		2.2	2.2	1.2	2.2
15	2.1	2.20	3.30	2.10	2.6	1.4	1.95	2.75	2.95		2.40	2.7	2.6	2.2	2.1	1.7	2.1	2.2	2.3		2.9		2.6	2.0	1.2	2.2
16	1.4	2.0	1.90	1.6	3.25	2.35	3.10	2.90	2.70		3.8	3.3	3.2	2.1	1.8	2.3	2.2	2.9		2.6		2.9	2.6	1.9	1.6
17	1.3	2.2 1.95	3.6	2.50	2.1	1.70	2.10	2.00		2.4	2.7	2.8	2.9	1.8	2.2	2.1	2.1		2.1		2.3	2.8	1.8	2.1
18	1.1	2.30		2.65	5.0	1.95	1.85		2.5	2.9	2.7	1.9		2.6		2.6			2.4	2.2	2.4
19	1.2	2.45			8.75			2.4	2.3	2.1		2.2		2.7				2.3
20	1.6								2.2	2.1		2.5						2.2

| X̄ | 1.4 | 2.1 | 2.6 | 2.5 | 2.7 | 2.7 | 2.5 | (2.9) | 2.2 | 2.4 | 2.8 | 2.6 | 2.9 | 2.8 | 2.1 | 2.5 | 2.5 | 2.8 | 3.0 | 2.8 | 2.5 | 2.6 | 2.6 | 2.4 | 2.1 | 2.2 |

↓

2.8   7/4 7/4 7/4

(If chamber #5 is 3.20 - please verify and recalculate if necessary).

① Chamber #2 only contained 15 specimens. 7/4

Chamber

 # Organisms

	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48
1	2.5	1.8	1.7	2.9	2.1	2.3	3.3	3.6	2.5	2.6	4.0	3.4	3.0	2.1	?.3	2.8	3.9	2.7	3.1	2.8	2.1	2.8	3.1
2	3.4	2.3	3.0	3.2	2.0	2.1	3.0	1.8	3.8	2.7	3.9	3.5	3.9	2.5	2.7	2.2	3.0	2.6	3.2	2.1	2.8	3.1	2.9
3	2.2	2.8	2.5	3.3	1.8	2.2	2.6	2.2	3.6	3.0	2.9	3.4	3.2	2.4	3.1	2.5	2.3	3.0	4.6	2.9	2.6	2.5	3.4
4	2.3	2.9	2.5	3.4	1.9	2.4	2.6	2.4	3.0	2.2	4.1	3.7	4.0	2.4	3.2	3.1	3.6	2.6	4.1	2.8	3.7	3.4	3.0
5	2.6	2.6	2.1	3.6	3.0	2.2	2.3	2.5	3.2	2.1	3.9	3.1	3.0	2.3	2.4	2.8	3.0	3.4	2.5	2.9	2.8	3.1	2.2
6	3.0	2.1	3.0	3.5	3.5	2.0	2.6	2.0	4.0	2.0	3.7	3.1	2.9	2.8	2.5	3.1	3.5	2.4	3.1	1.9	3.0	3.5	3.0
7	2.1	2.0	2.8	4.2	4.0	2.1	2.1	2.8	3.6	2.3	4.5	2.6	2.7	2.0	2.6	2.8	2.7	2.2	4.1	2.9	1.8	2.9	3.5
8	2.0	2.1	2.3	4.1	3.6	1.7	2.0	3.2	2.7	2.8	3.1	2.0	2.9	1.8	3.0	3.0	3.4	3.2	2.8	3.0	3.5	3.6	2.8
9	2.9	2.1	3.1	3.1	3.1	2.5	2.1	2.8	2.9	2.6	2.6	3.1	3.2	2.1	2.8	3.1	3.5	2.4	3.0	2.0	3.8	3.2	4.3
10	2.3	2.2	3.0	3.0	2.6	2.0	1.8	2.6	2.8	2.5	3.3	3.0	3.3	2.2	2.9	2.0	2.1	3.0	2.7	3.4	2.0	3.6	3.1
11	2.5	2.3	2.3	3.1	2.8	2.9	1.9	2.0	3.0	2.4	3.6	2.3	3.0	2.2	3.5	2.7	3.0	2.2	3.0	3.1	3.2	3.3	2.0
12	2.3	2.2	2.2	2.9	2.5	2.2	2.1	1.8	2.7	2.3	2.9	2.2	2.8	2.3	2.5	2.8	2.7	3.5	2.4	3.1	3.3	2.6	2.8
13	2.5	2.3	2.4	3.4	3.0	2.1	2.3	2.5	2.2	2.6	2.7	2.2	2.4	2.4	1.9	2.6	2.1	2.7	2.1	2.8	3.5	2.8	2.9
14	2.4	2.2	1.8	3.8	2.6	2.2	2.4	2.6	2.1	2.6	3.0	2.1	2.3	2.1	2.1	2.4	2.1	2.4	2.7	2.6	2.8	3.5	2.6
15	2.5	2.4	2.1	3.9	3.1	2.3	2.6		2.3	3.0	2.8	2.0	2.6	2.0	2.3	2.3	2.3	3.0	2.7	3.2	3.1	2.7	2.2
16	2.7	2.1	2.0	2.1	5.0	1.6	2.2		2.2	2.8	2.7	2.3	2.4		2.5	2.6	2.4	3.0	2.6	3.0	2.6	2.8	2.4
17		2.2	2.2	2.3	2.6		2.1		2.4	2.0	2.5	2.8	2.4		3.0		2.1		2.8	2.8	2.5	2.6	2.2
18			2.1	2.5	2.8		2.0				2.4	2.4	2.3		3.0				2.7	2.9	2.6		
19			2.3	2.9			1.8				2.5								2.9	2.9	2.6		
20				3.0			2.5												3.0	2.8			
X̄	2.5	1.3	2.4	3.3	2.8	2.2	2.3	2.5	2.9	2.5	3.2	2.6	2.9	2.2	2.7	2.7	2.8	3.0	2.8	2.9	3.1	2.8	

2.8

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chd SM 7/22/9

JD 7/23/97

97286sc
File: 97286SC   Transform: ARC SINE(SQUARE ROOT(Y))

Shapiro - Wilk's test for normality

D =   0.661

W =   0.989

Critical W (P = 0.05) (n = 48) = 0.947
Critical W (P = 0.01) (n = 48) = 0.929

Data PASS normality test at P=0.01 level. Continue analysis.

97286sc
File: 97286SC   Transform: ARC SINE(SQUARE ROOT(Y))

Bartlett's test for homogeneity of variance
Calculated B1 statistic =   8.87

Table Chi-square value =   18.48   (alpha = 0.01, df =   7)
Table Chi-square value =   14.07   (alpha = 0.05, df =   7)

Data PASS B1 homogeneity test at 0.01 level. Continue analysis.

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GRP	IDENTIFICATION	REP	VALUE	TRANS VALUE
1	CONTROL	1	0.8000	1.1071
1	CONTROL	2	0.8500	1.1731
1	CONTROL	3	0.8500	1.1731
1	CONTROL	4	0.8000	1.1071
1	CONTROL	5	0.8000	1.1071
1	CONTROL	6	0.9000	1.2490
2	A9-1	1	0.9500	1.3453
2	A9-1	2	1.0000	1.4588
2	A9-1	3	0.9500	1.3453
2	A9-1	4	0.8000	1.1071
2	A9-1	5	0.9000	1.2490
2	A9-1	6	0.9000	1.2490
3	A3-1	1	0.8500	1.1731
3	A3-1	2	0.7000	0.9912
3	A3-1	3	0.7000	0.9912
3	A3-1	4	0.8500	1.1731
3	A3-1	5	0.8500	1.1731
3	A3-1	6	0.6500	0.9377
4	A6-2	1	1.0000	1.4588
4	A6-2	2	0.8500	1.1731
4	A6-2	3	0.8500	1.1731
4	A6-2	4	0.8500	1.1731
4	A6-2	5	0.8500	1.1731
4	A6-2	6	0.9000	1.2490
5	A5-2	1	0.8500	1.1731
5	A5-2	2	0.7500	1.0472
5	A5-2	3	0.5500	0.8355
5	A5-2	4	0.9000	1.2490
5	A5-2	5	1.0000	1.4588
5	A5-2	6	0.9000	1.2490
6	A4-1	1	1.0000	1.4588
6	A4-1	2	0.9500	1.3453
6	A4-1	3	0.8500	1.1731
6	A4-1	4	1.0000	1.4588
6	A4-1	5	0.8500	1.1731
6	A4-1	6	0.8000	1.1071
7	A2-2	1	0.9500	1.3453
7	A2-2	2	0.8500	1.1731
7	A2-2	3	1.0000	1.4588
7	A2-2	4	0.9500	1.3453
7	A2-2	5	0.9500	1.3453
7	A2-2	6	0.9500	1.3453
8	A1-1	1	0.8500	1.1731
8	A1-1	2	0.9000	1.2490
8	A1-1	3	0.9000	1.2490
8	A1-1	4	0.8500	1.1731
8	A1-1	5	0.9500	1.3453
8	A1-1	6	1.0000	1.4588

97286sc
File: 97286SC   Transform: ARC SINE(SQUARE ROOT(Y))

SUMMARY STATISTICS ON TRANSFORMED DATA   TABLE 1 of 2

GRP	IDENTIFICATION	N	MIN	MAX	MEAN
1	CONTROL	6	1.107	1.249	1.153
2	A9-1	6	1.107	1.459	1.292
3	A3-1	6	0.938	1.173	1.073
4	A6-2	6	1.173	1.459	1.233
5	A5-2	6	0.835	1.459	1.169
6	A4-1	6	1.107	1.459	1.286
7	A2-2	6	1.173	1.459	1.335
8	A1-1	6	1.173	1.459	1.275

97286sc
File: 97286SC   Transform: ARC SINE(SQUARE ROOT(Y))

SUMMARY STATISTICS ON TRANSFORMED DATA   TABLE 2 of 2

GRP	IDENTIFICATION	VARIANCE	SD	SEM	C.V. %
1	CONTROL	0.003	0.057	0.023	4.96
2	A9-1	0.014	0.119	0.049	9.24
3	A3-1	0.012	0.111	0.045	10.35
4	A6-2	0.013	0.115	0.047	9.29
5	A5-2	0.045	0.211	0.086	18.06
6	A4-1	0.024	0.155	0.063	12.08
7	A2-2	0.008	0.092	0.037	6.86
8	A1-1	0.012	0.110	0.045	8.65

97286sc
File: 97286SC   Transform: ARC SINE(SQUARE ROOT(Y))

ANOVA TABLE

SOURCE	DF	SS	MS	F
Between	7	0.326	0.047	2.820
Within (Error)	40	0.661	0.017	
Total	47	0.988		

Critical F value =   2.25   (0.05,7,40)
Since F > Critical F REJECT Ho: All equal

97286sc
File: 97286SC   Transform: ARC SINE(SQUARE ROOT(Y))

        DUNNETT'S TEST   -   TABLE 1 OF 2   Ho:Control<Treatment
----

GROUP	IDENTIFICATION	TRANSFORMED MEAN	MEAN CALCULATED IN ORIGINAL UNITS	T STAT	SIG
1	CONTROL	1.153	0.833		
2	A9-1	1.292	0.917	-1.881	
3	A3-1	1.073	0.767	1.072	
4	A6-2	1.233	0.883	-1.086	
5	A5-2	1.169	0.825	-0.215	
6	A4-1	1.286	0.908	-1.795	
7	A2-2	1.335	0.942	-2.461	
8	A1-1	1.275	0.908	-1.643	

Dunnett table value = 2.42   (1 Tailed Value, P=0.05, df=40,7)

97286sc
File: 97286SC   Transform: ARC SINE(SQUARE ROOT(Y))

        DUNNETT'S TEST   -   TABLE 2 OF 2   Ho:Control<Treatment
----

GROUP	IDENTIFICATION	NUM OF REPS	Minimum Sig Diff (IN ORIG. UNITS)	% of CONTROL	DIFFERENCE FROM CONTROL
1	CONTROL	6			
2	A9-1	6	0.152	18.2	-0.083
3	A3-1	6	0.152	18.2	0.067
4	A6-2	6	0.152	18.2	-0.050
5	A5-2	6	0.152	18.2	0.008
6	A4-1	6	0.152	18.2	-0.075
7	A2-2	6	0.152	18.2	-0.108
8	A1-1	6	0.152	18.2	-0.075

97286SR
File: 97286SR   Transform: ARC SINE(SQUARE ROOT(Y))

Shapiro - Wilk's test for normality

D =   0.645

W =   0.989

Critical W (P = 0.05) (n = 42) = 0.942
Critical W (P = 0.01) (n = 42) = 0.922

Data PASS normality test at P=0.01 level. Continue analysis.

97286SR

Bartlett's test for homogeneity of variance
Calculated B1 statistic =   4.86

Table Chi-square value =   16.81   (alpha = 0.01, df =   6)
Table Chi-square value =   12.59   (alpha = 0.05, df =   6)

Data PASS B1 homogeneity test at 0.01 level. Continue analysis.

TITLE: 97286SR
FILE: 97286SR
TRANSFORM: ARC SINE(SQUARE ROOT(Y))   NUMBER OF GROUPS: 7

GRP	IDENTIFICATION	REP	VALUE	TRANS VALUE
1	A9-1	1	0.9500	1.3453
1	A9-1	2	1.0000	1.4588
1	A9-1	3	0.9500	1.3453
1	A9-1	4	0.8000	1.1071
1	A9-1	5	0.9000	1.2490
1	A9-1	6	0.9000	1.2490
2	A3-1	1	0.8500	1.1731
2	A3-1	2	0.7000	0.9912
2	A3-1	3	0.7000	0.9912
2	A3-1	4	0.8500	1.1731
2	A3-1	5	0.8500	1.1731
2	A3-1	6	0.6500	0.9377
3	A6-2	1	1.0000	1.4588
3	A6-2	2	0.8500	1.1731
3	A6-2	3	0.8500	1.1731
3	A6-2	4	0.8500	1.1731
3	A6-2	5	0.8500	1.1731
3	A6-2	6	0.9000	1.2490
4	A5-2	1	0.8500	1.1731
4	A5-2	2	0.7500	1.0472
4	A5-2	3	0.5500	0.8355
4	A5-2	4	0.9000	1.2490
4	A5-2	5	1.0000	1.4588
4	A5-2	6	0.9000	1.2490
5	A4-1	1	1.0000	1.4588
5	A4-1	2	0.9500	1.3453
5	A4-1	3	0.8500	1.1731
5	A4-1	4	1.0000	1.4588
5	A4-1	5	0.8500	1.1731
5	A4-1	6	0.8000	1.1071
6	A2-2	1	0.9500	1.3453
6	A2-2	2	0.8500	1.1731
6	A2-2	3	1.0000	1.4588
6	A2-2	4	0.9500	1.3453
6	A2-2	5	0.9500	1.3453
6	A2-2	6	0.9500	1.3453
7	A1-1	1	0.8500	1.1731
7	A1-1	2	0.9000	1.2490
7	A1-1	3	0.9000	1.2490
7	A1-1	4	0.8500	1.1731
7	A1-1	5	0.9500	1.3453
7	A1-1	6	1.0000	1.4588

97286SR
File: 97286SR   Transform: ARC SINE(SQUARE ROOT(Y))

SUMMARY STATISTICS ON TRANSFORMED DATA   TABLE 1 of 2

GRP	IDENTIFICATION	N	MIN	MAX	MEAN
1	A9-1	6	1.107	1.459	1.292
2	A3-1	6	0.938	1.173	1.073
3	A6-2	6	1.173	1.459	1.233
4	A5-2	6	0.835	1.459	1.169
5	A4-1	6	1.107	1.459	1.286
6	A2-2	6	1.173	1.459	1.335
7	A1-1	6	1.173	1.459	1.275

97286SR
File: 97286SR   Transform: ARC SINE(SQUARE ROOT(Y))

SUMMARY STATISTICS ON TRANSFORMED DATA   TABLE 2 of 2

GRP	IDENTIFICATION	VARIANCE	SD	SEM	C.V. %
1	A9-1	0.014	0.119	0.049	9.24
2	A3-1	0.012	0.111	0.045	10.35
3	A6-2	0.013	0.115	0.047	9.29
4	A5-2	0.045	0.211	0.086	18.06
5	A4-1	0.024	0.155	0.063	12.08
6	A2-2	0.008	0.092	0.037	6.86
7	A1-1	0.012	0.110	0.045	8.65

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97286SR
File: 97286SR   Transform: ARC SINE(SQUARE ROOT(Y))

ANOVA TABLE

SOURCE	DF	SS	MS	F
Between	6	0.289	0.048	2.609
Within (Error)	35	0.645	0.018	
Total	41	0.934		

Critical F value =   2.42   (0.05,6,30)
Since F > Critical F   REJECT   Ho: All equal

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97286SR
File: 97286SR   Transform: ARC SINE(SQUARE ROOT(Y))

DUNNETT'S TEST   -   TABLE 1 OF 2   Ho:Control<Treatment

GROUP	IDENTIFICATION	TRANSFORMED MEAN	MEAN CALCULATED IN ORIGINAL UNITS	T STAT	SIG
1	A9-1	1.292	0.917		
2	A3-1	1.073	0.767	2.797	*
3	A6-2	1.233	0.883	0.754	
4	A5-2	1.169	0.825	1.578	
5	A4-1	1.286	0.908	0.082	
6	A2-2	1.335	0.942	-0.550	
7	A1-1	1.275	0.908	0.226	

Dunnett table value =   2.40   (1 Tailed Value, P=0.05,   df=30,6)

97286SR
File: 97286SR   Transform: ARC SINE(SQUARE ROOT(Y))

DUNNETT'S TEST   -   TABLE 2 OF 2   Ho:Control<Treatment

GROUP	IDENTIFICATION	NUM OF REPS	Minimum Sig Diff (IN ORIG. UNITS)	% of CONTROL	DIFFERENCE FROM CONTROL
1	A9-1	6			
2	A3-1	6	0.127	13.8	0.150
3	A6-2	6	0.127	13.8	0.033
4	A5-2	6	0.127	13.8	0.092
5	A4-1	6	0.127	13.8	0.008
6	A2-2	6	0.127	13.8	-0.025
7	A1-1	6	0.127	13.8	0.008

97-286
File: 97286c   Transform: NO TRANSFORMATION

Shapiro - Wilk's test for normality

D =   2.700

W =   0.963

Critical W (P = 0.05) (n = 48) = 0.947
Critical W (P = 0.01) (n = 48) = 0.929

Data PASS normality test at P=0.01 level. Continue analysis.

97-286
File: 97286c   Transform: NO TRANSFORMATION

Bartlett's test for homogeneity of variance
Calculated B1 statistic =   2.29

Table Chi-square value =   18.48   (alpha = 0.01, df =   7)
Table Chi-square value =   14.07   (alpha = 0.05, df =   7)

Data PASS B1 homogeneity test at 0.01 level. Continue analysis.

```
TITLE:        97-286
FILE:         97286c
TRANSFORM: NO TRANSFORMATION                    NUMBER OF GROUPS: 8
-------------------------------------------------------------------------------

GRP  IDENTIFICATION    REP        VALUE           TRANS VALUE
---  --------------    ----     ------------      ------------
 1       control        1        2.8000            2.8000
 1       control        2        2.8000            2.8000
 1       control        3        2.3000            2.3000
 1       control        4        2.7000            2.7000
 1       control        5        2.5000            2.5000
 1       control        6        2.8000            2.8000
 2         A9-1         1        3.0000            3.0000
 2         A9-1         2        2.2000            2.2000
 2         A9-1         3        2.1000            2.1000
 2         A9-1         4        2.4000            2.4000
 2         A9-1         5        2.4000            2.4000
 2         A9-1         6        2.1000            2.1000
 3         A3-1         1        2.8000            2.8000
 3         A3-1         2        2.5000            2.5000
 3         A3-1         3        2.8000            2.8000
 3         A3-1         4        2.6000            2.6000
 3         A3-1         5        2.6000            2.6000
 3         A3-1         6        3.0000            3.0000
 4         A6-2         1        2.9000            2.9000
 4         A6-2         2        2.5000            2.5000
 4         A6-2         3        3.1000            3.1000
 4         A6-2         4        2.5000            2.5000
 4         A6-2         5        2.5000            2.5000
 4         A6-2         6        2.9000            2.9000
 5         A5-2         1        2.2000            2.2000
 5         A5-2         2        2.2000            2.2000
 5         A5-2         3        2.6000            2.6000
 5         A5-2         4        2.7000            2.7000
 5         A5-2         5        2.8000            2.8000
 5         A5-2         6        2.6000            2.6000
 6         A4-1         1        2.3000            2.3000
 6         A4-1         2        2.5000            2.5000
 6         A4-1         3        2.8000            2.8000
 6         A4-1         4        2.1000            2.1000
 6         A4-1         5        2.5000            2.5000
 6         A4-1         6        2.2000            2.2000
 7         A2-2         1        2.9000            2.9000
 7         A2-2         2        2.7000            2.7000
 7         A2-2         3        3.3000            3.3000
 7         A2-2         4        2.4000            2.4000
 7         A2-2         5        2.8000            2.8000
 7         A2-2         6        2.9000            2.9000
 8         A1-1         1        2.5000            2.5000
 8         A1-1         2        2.6000            2.6000
 8         A1-1         3        2.7000            2.7000
 8         A1-1         4        2.9000            2.9000
 8         A1-1         5        3.2000            3.2000
 8         A1-1         6        2.8000            2.8000
-------------------------------------------------------------------------------
```

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SUMMARY STATISTICS ON TRANSFORMED DATA   TABLE 1 of 2

GRP	IDENTIFICATION	N	MIN	MAX	MEAN
1	control	6	2.300	2.800	2.650
2	A9-1	6	2.100	3.000	2.367
3	A3-1	6	2.500	3.000	2.717
4	A6-2	6	2.500	3.100	2.733
5	A5-2	6	2.200	2.800	2.517
6	A4-1	6	2.100	2.800	2.400
7	A2-2	6	2.400	3.300	2.833
8	A1-1	6	2.500	3.200	2.783

97-286
File: 97286c   Transform: NO TRANSFORMATION

SUMMARY STATISTICS ON TRANSFORMED DATA   TABLE 2 of 2

GRP	IDENTIFICATION	VARIANCE	SD	SEM	C.V. %
1	control	0.043	0.207	0.085	7.83
2	A9-1	0.115	0.339	0.138	14.31
3	A3-1	0.034	0.183	0.075	6.75
4	A6-2	0.071	0.266	0.109	9.73
5	A5-2	0.066	0.256	0.105	10.18
6	A4-1	0.064	0.253	0.103	10.54
7	A2-2	0.087	0.294	0.120	10.39
8	A1-1	0.062	0.248	0.101	8.92

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7/24

Transform: NO TRANSFORMATION

ANOVA TABLE

SOURCE	DF	SS	MS	F
Between	7	1.310	0.187	2.772
Within (Error)	40	2.700	0.067	
Total	47	4.010		

Critical F value =   2.25   (0.05,7,40)
Since F > Critical F   REJECT   Ho: All equal

DUNNETT'S TEST   —   TABLE 1 OF 2   Ho:Control<Treatment

GROUP	IDENTIFICATION	TRANSFORMED MEAN	MEAN CALCULATED IN ORIGINAL UNITS	T STAT	SIG
1	control	2.650	2.650		
2	A9-1	2.367	2.367	1.889	
3	A3-1	2.717	2.717	-0.444	
4	A6-2	2.733	2.733	-0.556	
5	A5-2	2.517	2.517	0.889	
6	A4-1	2.400	2.400	1.667	
7	A2-2	2.833	2.833	-1.222	
8	A1-1	2.783	2.783	-0.889	

Dunnett table value = 2.42   (1 Tailed Value, P=0.05, df=40,7)

DUNNETT'S TEST   —   TABLE 2 OF 2   Ho:Control<Treatment

GROUP	IDENTIFICATION	NUM OF REPS	Minimum Sig Diff (IN ORIG. UNITS)	% of CONTROL	DIFFERENCE FROM CONTROL
1	control	6			
2	A9-1	6	0.363	13.7	0.283
3	A3-1	6	0.363	13.7	-0.067
4	A6-2	6	0.363	13.7	-0.083
5	A5-2	6	0.363	13.7	0.133
6	A4-1	6	0.363	13.7	0.250
7	A2-2	6	0.363	13.7	-0.183
8	A1-1	6	0.363	13.7	-0.133

97-286
File: 97286r   Transform: NO TRANSFORMATION

Shapiro - Wilk's test for normality

D =   2.317

W =   0.955

Critical W (P = 0.05) (n = 36) = 0.935
Critical W (P = 0.01) (n = 36) = 0.912

Data PASS normality test at P=0.01 level. Continue analysis.

97-286
File: 97286r   Transform: NO TRANSFORMATION

Bartlett's test for homogeneity of variance
Calculated B1 statistic =   0.71

Table Chi-square value =   15.09   (alpha = 0.01, df =   5)
Table Chi-square value =   11.07   (alpha = 0.05, df =   5)

Data PASS B1 homogeneity test at 0.01 level. Continue analysis.

GRP	IDENTIFICATION	REP	VALUE	TRANS VALUE
1	Reference A9-1	1	3.0000	3.0000
1	Reference A9-1	2	2.2000	2.2000
1	Reference A9-1	3	2.1000	2.1000
1	Reference A9-1	4	2.4000	2.4000
1	Reference A9-1	5	2.4000	2.4000
1	Reference A9-1	6	2.1000	2.1000
2	A6-2	1	2.9000	2.9000
2	A6-2	2	2.5000	2.5000
2	A6-2	3	3.1000	3.1000
2	A6-2	4	2.5000	2.5000
2	A6-2	5	2.5000	2.5000
2	A6-2	6	2.9000	2.9000
3	A5-2	1	2.2000	2.2000
3	A5-2	2	2.2000	2.2000
3	A5-2	3	2.6000	2.6000
3	A5-2	4	2.7000	2.7000
3	A5-2	5	2.8000	2.8000
3	A5-2	6	2.6000	2.6000
4	A4-1	1	2.3000	2.3000
4	A4-1	2	2.5000	2.5000
4	A4-1	3	2.8000	2.8000
4	A4-1	4	2.1000	2.1000
4	A4-1	5	2.5000	2.5000
4	A4-1	6	2.2000	2.2000
5	A2-2	1	2.9000	2.9000
5	A2-2	2	2.7000	2.7000
5	A2-2	3	3.3000	3.3000
5	A2-2	4	2.4000	2.4000
5	A2-2	5	2.8000	2.8000
5	A2-2	6	2.9000	2.9000
6	A1-1	1	2.5000	2.5000
6	A1-1	2	2.6000	2.6000
6	A1-1	3	2.7000	2.7000
6	A1-1	4	2.9000	2.9000
6	A1-1	5	3.2000	3.2000
6	A1-1	6	2.8000	2.8000

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SUMMARY STATISTICS ON TRANSFORMED DATA   TABLE 1 of 2

GRP	IDENTIFICATION	N	MIN	MAX	MEAN
1	Reference A9-1	6	2.100	3.000	2.367
2	A6-2	6	2.500	3.100	2.733
3	A5-2	6	2.200	2.800	2.517
4	A4-1	6	2.100	2.800	2.400
5	A2-2	6	2.400	3.300	2.833
6	A1-1	6	2.500	3.200	2.783

SUMMARY STATISTICS ON TRANSFORMED DATA   TABLE 2 of 2

GRP	IDENTIFICATION	VARIANCE	SD	SEM	C.V. %
1	Reference A9-1	0.115	0.339	0.138	14.31
2	A6-2	0.071	0.266	0.109	9.73
3	A5-2	0.066	0.256	0.105	10.18
4	A4-1	0.064	0.253	0.103	10.54
5	A2-2	0.087	0.294	0.120	10.39
6	A1-1	0.062	0.248	0.101	8.92

97-286
File: 97286r   Transform: NO TRANSFORMATION

ANOVA TABLE

SOURCE	DF	SS	MS	F
Between	5	1.242	0.248	3.217
Within (Error)	30	2.317	0.077	
Total	35	3.559		

Critical F value =   2.53   (0.05,5,30)
Since F > Critical F   REJECT   Ho: All equal

DUNNETT'S TEST   -   TABLE 1 OF 2   Ho:Control<Treatment

GROUP	IDENTIFICATION	TRANSFORMED MEAN	MEAN CALCULATED IN ORIGINAL UNITS	T STAT	SIG
1	Reference A9-1	2.367	2.367		
2	A6-2	2.733	2.733	-2.285	
3	A5-2	2.517	2.517	-0.935	
4	A4-1	2.400	2.400	-0.208	
5	A2-2	2.833	2.833	-2.909	
6	A1-1	2.783	2.783	-2.597	

Dunnett table value = 2.33   (1 Tailed Value, P=0.05,   df=30,5)

DUNNETT'S TEST   -   TABLE 2 OF 2   Ho:Control<Treatment

GROUP	IDENTIFICATION	NUM OF REPS	Minimum Sig Diff (IN ORIG. UNITS)	% of CONTROL	DIFFERENCE FROM CONTROL
1	Reference A9-1	6			
2	A6-2	6	0.374	15.8	-0.367
3	A5-2	6	0.374	15.8	-0.150
4	A4-1	6	0.374	15.8	-0.033
5	A2-2	6	0.374	15.8	-0.467
6	A1-1	6	0.374	15.8	-0.417

APPENDIX E

Hazard Characterization
Cornell-Dubilier Site
South Plainfield, NJ
April 1998

APPENDIX E

HAZARD CHARACTERIZATION

E.1   VOAs

E.1.1   Trans-1,2-Dichloroethene

Trans-1,2-Dichloroethenes are commonly found in landfills, aquifers, and sediment as breakdown products from the reductive halogenation of common industrial solvents trichloroethylene, tetrachloroethylene, and 1,1,2,2-tetrachloroethane. The isomer cis-1,2,-dichloroethylene is more common than the trans-isomer and is often mistakenly reported as the trans-isomer. Releases of trans-1,2-Dichloroethene to soil should result in evaporation and leaching into ground water whereupon it may slowly biodegrade. If released into the water, it will be lost mainly through volatilization. Biodegradation and adsorption to sediments is not considered significant. Trans-1,2-Dichloroethene is not considered to bioconcentrate significantly in aquatic organisms. A bioconcentration factor of 22 has been estimated for this compound using a recommended octanol/water partition coefficient of 2.06 (Howard 1990).

E.1.2   1,1,2,2-Tetrachloroethane

1,1,2,2-Tetrachloroethane in the environment is extremely stable. Volatilization over the course of days to weeks removes most of this compound in surface water. It does not partition itself to the organic constituent in sediment nor to suspended solids. It is predicted to be highly mobile in soil, and thus, to leach into the ground water. There is some evidence that this compound slowly biodegrades, and under alkaline conditions, to hydrolyze. 1,1,2,2-Tetrachloroethane does not bioconcentrate. An experimental log bioconcentration factor of 0.9 for the bluegill sunfish was reported after a 14-day exposure to an ambient concentration of 9.62 $\mu g/L$ (Howard 1990). If ingested, this compound is readily absorbed by the gastrointestinal tract (Clayton and Clayton 1981-1982). Approximately half of the 1,1,2,2-tetrachlorethane administered to mice was expired as CO_2. Dichloroacetic acid, oxalic acid, and glyoxylic acid was formed and excreted in the urine. The remainder was metabolized as glycine (The Chemical Society 1972).

E.1.3   1,1,1-Trichloroethane

1,1,1-Trichloroethane enters the environment through air emissions, in wastewater and from its production, or from use in vapor degreasing and metal cleaning. Releases of this compound to water will result in almost complete removal through evaporation. Biodegradation and adsorption to sediment are considered insignificant. In soil, 1,1,1-trichloroethane volatilizes into the atmosphere and percolates into the ground water. This compound has little tendency to bioconcentrate in aquatic organisms. One experimental study reported a bioconcentration factor of 8.9 for bluegill sunfish in a 28-day test (Howard 1990). Trichloroethane is rapidly absorbed through the lungs and gastrointestinal tract, but cutaneous absorption is too slow to produce significant toxicity (Ellenhorn and Barceloux 1988).

In the mammalian body, a small percentage of 1,1,1-trichloroethane is metabolized to carbon dioxide, while the remainder appears in urine as glucoronide of 2,2,2-trichloroethanol (ILO 1983).

E.1.4   1,1,2-Trichloroethane

1,1,2-Trichloroethane enters the environment through the manufacture of vinylidene chloride (cis-

1,2-dichlorothene) and its use as a solvent. Evaporation removes most of the 1,1,2-trichloroethane from surface water. This compound has a low soil partition coefficient and therefore will not partition into sediment and will readily pass through soil into the ground water. Biodegradation in ground water and bioconcentration in aquatic organisms is not likely to be significant (Howard 1990).

E.1.5 Trichlorofluoromethane

Trichlorofluoromethane, also referred to as Freon 11, was primarily released to the environment during its use as an aerosol propellant. Although this use was banned in 1978, it is currently used as a refrigerant, foaming agent for polyurethane foams, solvent and degreaser, and fire extinguishing agent. Because of its high vapor pressure and environmental stability, trichlorofluoromethane volatilizes rapidly from water and soil, and may leach into the ground water where it may persist for a long time. Biodegradation and adsorption of this compound to sediment is considered to be insignificant (Howard 1990). This compound does not readily accumulate and if absorbed, is usually rapidly eliminated from the body (ACGIH 1986). The theoretical metabolites of trichlorofluoromethane are dichlorofluoromethane and tetrachlorofluoromethane (NRC 1980).

E.1.6 Acetone

Acetone is one of the least hazardous industrial solvents, but it is highly volatile. It is also released naturally from volcanoes and forest fires, is a natural product of plant and animal metabolism. Acetone on soil will volatilize and leache into the ground water, whereupon it may biodegrade. Biodegradation and volatilization occurs to acetone in water. Bioconcentration in aquatic organisms and adsorption to sediment is not considered to be significant. One experimental study reported a bioconcentration factor of 0.69 for adult haddock at 7 to 9°C in a static system.

E.1.7 Carbon Disulfide

Carbon disulfide is a natural product of anaerobic biodegradation and also arises from geothermal sources. It may be released as emissions and in wastewater during its production and use, from the production of viscose rayon, cellophane, and carbon tetrachloride. Carbon disulfide volatilizes readily from soil and may also readily leach into the ground water, whereupon it may biodegrade. Volatilization is also the primary means of removal from water; bioconcentration by aquatic organisms and adsorption to sediment is not considered to be significant (Howard 1990).

In the organism, carbon disulfide reacts with a variety of nucleophilic functional groups to form dithiocarbamic acids, trithiocarbamic acids, xanthogenic acids, and heterocycles. A small amount of carbon disulfide is converted to hydrogen sulfide, which is rapidly oxidized to sulfate and excreted in the urine. Carbon disulfide is recognized as an inhibitor of brain monoamine oxidase through two possible mechanisms (Gosselin et al. 1984).

E.1.8 Chloromethane

Chloromethane, also commonly referred to as methyl chloride, occurs naturally in oceans, from forest and brush fires, and from volcanoes. Anthropogenic sources of this compound are significant, and arise from its production and use in the manufacture of silicones and other chemicals, and as a solvent and propellant. Chloromethane volatilizes rapidly from water and soil, although in soil there is the potential for leaching into ground water. Once in the ground water, this compound biodegrades and hydrolyzes very slowly. Chloromethane has a very low log octanol/water partition coefficient, suggesting that it does not bioconcentrate to any appreciable degree in aquatic organisms (Howard 1989). In the mammalian body, chloromethane is broken down into methanol and hydrochloric acid.

The speed and extent of this breakdown is not known. The methanol resulting from this reaction is subsequently oxidized to formaldehyde (U.S. EPA 1978).

The primary effect of chloromethane is cytotoxicity through disruption of cell metabolism and altered electron transport processes of the respiratory chain (Mamedov and Aliev 1986).

E.1.9   1,1-Dichloroethylene; 1,1-Dichloroethene; Vinylidene Chloride

1,1-Dichloroethene, commonly referred to as 1,1-Dichloroethene or vinylidene chloride, enters the environment from its production and use in the manufacture of plastics such as kitchen plastic wrap. This compound in water will primarily be lost through evaporation. Little of it will adsorb to sediment. Once in the atmosphere, it degrades rapidly by photooxidation. 1,1-Dichloroethene on soil surfaces are lost through evaporation and partially by percolation into the ground water. In the ground water, very slow hydrolysis and biodegradation occurs. No experimental data has currently been found suggesting the potential for 1,1-Dichloroethene to bioconcentrate. The low octanol/water partition coefficient suggests that bioconcentration would not occur to any significant degree (Howard 1989).

E.1.10   Methylene Chloride (Dichloromethane)

Methylene chloride, commonly referred to as dichloromethane or DCM, is used widely and in large amounts in aerosols, paint removers, and in chemical processing. Most of this methylene chloride is released to the atmosphere where it degrades by reaction with photochemically produced hydroxyl radicals. Direct photolysis does not occur with this compound. Releases to water are primarily removed by evaporation. Hydrolysis in water is not considered to be an important removal process. Biodegradation may occur but would probably be very slow compared to evaporation. It is not expected to significantly adsorb to sediment or to bioconcentration in aquatic organisms. Due to its high vapor pressure, methylene chloride that is released to soil evaporates rapidly from near the soil surface. It is probable that a portion of a release will leach into the ground water. The mechanism of degradation in ground water is unknown for this compound. Hydrolysis in soil or ground water is probably not an important process under normal environmental conditions. Although experimental data is lacking, methylene chloride is not expected to bioconcentrate due to its low octanol/water partition coefficient from which a bioconcentration factor of 5 has been calculated (Howard 1990).

E.1.11   Toluene

Toluene enters the environment principally from the volatilization of petroleum fuels, toluene-based solvents and thinners, and motor vehicle exhaust. Toluene in soil near the soil surface will be lost through evaporation with some leaching into the ground water. Biodegradation occurs slowly in soil and ground water, especially at high concentrations, at which toluene may be toxic to microorganisms. However, acclimated microorganisms may biodegrade toluene rapidly. It does not hydrolyze significantly in soil or water. Toluene in water is lost by volatilization and biodegradation, the rate of which depends strongly on temperature, mixing conditions, and the existence of acclimated microorganisms. The half-life ranges from days to several weeks. Toluene does not significantly hydrolyze, directly photolyze, adsorb to sediment, or bioconcentrate in aquatic organisms. Some bioconcentration factors in aquatic organisms are as follows: eel (*Anguilla japonica*), 13.2; Manila clam (*Tapes semidecussata*), 1.67; blue mussel (*Mytilus edulis*), 4.2; green algae (*Chlorella fusca*), 380; golden ide fish (*Leuciscus idus melanotus*), 90 (Howard 1990).

Toluene in solution is readily absorbed by the gastrointestinal tract but is poorly absorbed through the skin (Browning 1965). Low levels of toluene (less than 100 parts per million) may produce disturbances in dopaminergic mechanisms of the basal ganglia, probably leading to functional

E-3

changes in sensory-motor integration (Fuxe et al. 1982). Exposure to toluene causes both reversible and irreversible changes to the central nervous system (Bjornaes and Naalsund 1988).

E.1.12   Other VOAs

Information on the fate, transport, general chemistry and toxicology of the remaining VOAs evaluated in this risk assessment could not be located at the time of this report.

E.2   BNAs

E.2.1   Phthalates

E.2.1.1   Bis(2-ethylhexyl) phthalate

Bis(2-ethylhexyl) phthalate is used in plasticizing, as an organic pump fluid, as a compouent of dielectric fluids, and as a solvent in insect repellant formulations, cosmetics, rubbing alcohol, liquid soap, detergents, decorative inks, lacquers, munitions, industrial and lubricating oils, defoaming agents during paper and paperboard manufacture, and as a pesticide carrier (HSDB 1997).

If released to soil, bis(2-ethylhexyl) phthalate will neither evaporate nor leach into groundwater, as it adsorbs strongly to soil. Limited data is available to suggest that it might biodegrade in soil under aerobic conditions following acclimation. In aquatic environments, bis(2-ethylhexyl) phthalate will biodegrade rapidly under aerobic conditions, with a half-life of two to three weeks. It will strongly adsorb to sediment, and evaporation and hydrolysis are not significant removal processes (HSDB 1997).

The four primary metabolic processes that bis(2-ethylhexyl) phthalate undergoes include hydrolysis of the ester, cleavage of the benzene ring, oxidation of the liberated alcohol, and oxidation of the alkyl side-chain while bound to the phthalate ester. In rats, metabolites include 5-keto-2-ethylhexyl phthalate, 5-carboxyl-2-ethylpentyl phthalate, 5-hydroxy-2-ethylhexyl phthalate, and 2-carboxymethylbutyl phthalate after initial hydrolysis to mono(2-ethylhexyl) phthalate. In green monkeys and ferrets, metabolites include the glucuronide derivatives of mono-(2-ethylhexyl) phthalate. In fish, liver homogenates metabolized the parent compound to monoethylhexyl phthalate. Fathead minnows and rainbow trout were also found to produce this compound as well as its glucuse conjugate. It has been shown in mammals that the mono-(2-ethylhexyl) phthalate is the intermediate that is responsible for producing degenerated spermatocytes. The observed teratogencity of bis(2-ethylhexyl) phthalate is believed to be due to the hydrolysis of the parent compound to 2-ethylhexanol, which in turn is metabolized to 2-ethylhexanoic acid, the proximate teratogen (HSDB 1997).

Mammals exhibit extremely low acute toxicity as a result of oral exposure to bis(2-ethylhexyl) phthalate. The oral LD50s in rats, mice, rabbits, and Guinea pigs have been calculated to be 30,000 mg/kg, 1500 mg/kg, 34,000 mg/kg, and 26,000 mg/kg, respectively. A variety of chronic effects have also been observed as a result of oral exposure to this contaminant. In dogs, effects on the liver were observed, including fatty vacuolization and congested areas of the liver, and liver function tests were negative. Lipid metabolism also appears to be inhibited by this contaminant, and this effect is transmitted across the palcental barrier to the developing fetus. Other development effects of bis(2-ethylhexyl) phthalateinclude suppression of maternal weight gain, increased fetal resorptions, neural tube defects, intrauterine growth retardation and delayed ossification. Seminiferous tubular degeneration, testicular degeneration, hypertrophy of cells in the anterior pituitary, as well as

hepatocellular carcinomas are some other effects which have been observed in mammals as a result of oral exposure to bis(2-ethylhexyl) phthalate (HSDB 1997).

In the aquatic environment, *Daphnia magna* have exhibited decreased reproduction at concentrations of 3, 10, and 30 ug/L, while the 48-hour LC50 for this organism has been reported to be between 1000 and 5000 ug/L. The LC50s of two other invertebrate species, *Chironomus plumosus* (midge) and *Gammarus pseudolimnaeus* (scud), are reported to be > 18 mg/L and > 32 mg/L, respectively. In fish, LC50s are reported to be greater than 100 mg/L for five species tested. The experimental BCFs for bis(2-ethylhexyl) phthalate range from a log of 1 to 4 in fish and invertebrates (HSDB 1997).

No significant toxic effects have been observed in birds as a result of oral exposure to bis(2-ethylhexyl) phthalate. However, this is probably due to the fact that this contaminant has not been studied extensively in birds (HSDB 1997).

E.2.1.2 Butylbenzyl phthalate

Butlylbenzyl phthalate is used as an organic intermediate, a plasticizer, and in coatings. When released to soil, it is expected to adsorb strongly and thus not to migrate significantly into the groundwater, although it has been detected in some groundwater samples. It is readily biodegraded in a variety of media under both aerobic and anaerobic conditions. When released into the aquatic environment, butylbenzyl phthalate will adsorb strongly to sediments. It is not expected to undergo abiotic degradation processes, such as photodegradation and hydrolysis, to a significant degree (HSDB 1997).

Butylbenzyl phthalate undergoes metabolic reactions similar to bis(2-ethylhexyl) phthalate, described previously, with the monophthalate metabolic products being the most abundant. It is believed that the mode of acute toxicity for butylbenzyl phthalate may be through its effects on the catecholamines of the central adrenergic nervous system (HSDB 1997).

In mammals, butylbenzyl phthalate has been shown to produce a variety of adverse toxicological effects, including central and peripheral neuropathies, increased incidence of myelomonocytic leukemia, thymic atrophy, splenitis, and atrophy of the testes, prostate, and epididymis. The oral LD50 in rats was calculated to be 13,500 mg/kg (HSDB 1997).

In the aquatic environment, butylbenzyl phthalate is acutely toxic to a variety of algae, invertebrates, and fish in the 0.5 to 5.0 mg/L range and chronically toxic to *Daphnia* and fathead minnows in the 0.1 to 0.8 mg/L range. For example, the 48-hour EC50 value for *Daphnia magna* was found to be 1.0 mg/L. In fish, butylbenzyl phthalate decreased the heart rate of goldfish at 200 mg/L, although this effect may have been due to dibutyl phthalate contamination. The 96-hour LC50 value for English sole is between 0.55 and 0.66 mg/L, and for bluegill is 43 mg/L. The bioconcentration factor (BCF) for a bluegill sunfish was calculated to be 663, and another source states that butylbenzyl phthalate is not an accumulative or persistent chemical in fish (HSDB 1997).

In birds, limited studies have been performed to assess the toxicity of butylbenzyl phthalate. In one study, no malformations were observed when butylbenzyl phthalate was injected into fertilized hens' eggs. In another study, it was found that butylbenzyl phthalate had an ED50 for embryotoxicity of approximately 27 umol/egg (HSDB 1997).

E.2.1.3 Diethyl phthalate

Diethyl phthalate is used as a solvent, fixative, wetting agent, plasticizer, camphor substitute, and in insecticidal sprays and mosquito repellents. If released to soil, diethyl phthalate is expected to undergo aerobic biodegradation. Anaerobic biodegradation may be slow or non-existent. Oxidation, chemical hydrolysis, and volatilization from wet soil surfaces are expected to be minimal, but evidence suggests that volatilization from dry surfaces may occur. If released into the aquatic environment, aerobic biodegradation is expected to occur, resulting in an approximate half-life of two days to greater than two weeks. As in soil, anaerobic biodegradation, chemical hydrolysis, oxidation, and volatilization are not expected to occur to a significant degree, expect that volatilization may occur in shallow water bodies. Photolysis should also not be significant. Diethyl phthalate will adsorb to suspended solids, thus providing a transport mechanism in the aquatic environment. However, it was observed that diethyl phthalate did not adsorb significantly to sediments during a study using this chemical in an aqueous environment (HSDB 1997).

The metabolic pathway of diethyl phthalate is similar to that of the other phthalates, discussed previously. In mammals, its metabolism includes transformation to its half-esters, which have been found to be four times more toxic than the parent compound. A potential mechanism of toxicity has been elucidated in that diethyl phthalate and dimethoxyethyl phthalate inhibited UDP-glucuronyltransferase activity of rat liver in vitro (HSDB 1997).

The LD50 for diethyl phthalate in rats is 9,000 mg/kg. Diethyl phthalate has been shown to produce a reduction in food intake and in the rate of body weight gain. It has also been shown to decrease testosterone concentrations in the testes. Tests in rats for teratogenicity have been negative (HSDB 1997).

In the aquatic environment, diethyl phthalate is slightly toxic to a variety of organisms. The 96-hour LC50 for the copepod *Nitocra spinipes* was found to be 74 mg/L. In fish, the 96-hour LC50s for sheepshead minnow and bluegill sunfish were 30 mg/L and 110 mg/L, respectively. It has been shown that diethyl phthalate will bioaccumulate in some aquatic organisms, but will not tend to biomagnify because they are metabolized relatively rapidly by fish and other animals. The bioconcentration factor (BCF) has been measured in bluegill sunfish and Mullet, and was calculated to be 117 and 15-16, respectively (HSDB 1997).

No information could be found at the time of this report on the effects of diethyl phthalate in birds.

E.2.1.4 Dimethyl phthalate

Dimethyl phthalate is used as a solvent and a plasticizer, and is used in insect repellants, varnishes, clear films, solid rocket propellants, lacquers, perfumes, and cosmetics. If released onto soil, dimethyl phthalate will likely migrate into the groundwater because it does not adsorb strongly to soil. It is readily biodegraded, especially after a short period of acclimation. In freshwater systems, dimethyl phthalate also biodegrades rapidly, with a half-life of less than eleven days in river water. Biodegradation is much slower in salt water systems. Volatilization may be significant in shallow water and photolysis may be a significant removal mechanism in clear water. Dimethyl phthalate only weakly adsorbs to sediment, and thus will tend to partition to the aqueous phase. Chemical hydrolysis may also occur under alkaline conditions (HSDB 1997).

Dimethyl phthalate is metabolized similarly to the other phthalates, discussed previously. In rats, it is primarily biotransformed iont monomethyl phthalate. Its mechanism of toxicity involves inhibition of mitochondrial respiration in the presence of succinate and adenosine diphosphate (ADP). It has also been shown to weakly inhibit lecithin/cholesterol acyltransferase (HSDB 1997).

In mammals, dimethyl phthalate exhibits a very low degree of acute toxicity. Acute LD50s have been reported to be 2400 to 6800 mg/kg for the rat, 6800 to 7200 mg/kg for the mouse, 2400 mg/kg for the Guinea pig, and greater than 1400 mg/kg for the dog. The results of most subchronic and chronic tests indicate that dimethyl phthalate is relatively nontoxic. Some of the toxic effects that have been observed as a result of oral exposure to dimethyl phthalate are slight nephritic changes, increase in respiratory rate, a decrease in testosterone on the testes and serum, and a decrease in total liver cholesterol and total liver lipids (HSDB 1997).

Very limited information is available on the toxicity of dimehtyl phthalate to fish and aquatic invertebrates. However, 96-hour EC50s have been reported for a variety of algal species, and they range from 26.1 mg/L for *Skeletonema costatum* to 125 mg/L for *Gymnodinium breve*. Dimethyl phthalate is not expected to bioconcentrate in fish. The mean measure BCFs for brown shrimp and sheepshead minnow ranged from 4.7 to 5.4 after 24 hours. A slightly higher BCF of 57 was measured for the bluegill sunfish, which may be explained by the fact that only carbon-14 was measured in the experiment, and thus metabolites might have been included in the measurements for the parent compound (HSDB 1997). A biomagnification factor of 130 was also calculated for edible fish (OHM/TADS 1997).

In birds, an oral LD50 of 10,115 mg/kg has been reported for the chicken. In addition, when chick embryos were exposed to chick ringers solution saturated with dimethyl phthalate, growth retardation and CNS malformations were observed (HSDB 1997).

E.2.1.5   Di-n-butyl phthalate

Di-n-butyl phthalate is used as an insect repellant, solvent, plasticizer, and a reaction media for chemical reactions. It is also used in explosives, nail polish, perfumes, printing inks, paper coatings, adhesives, medications and rocket propellants. If released into soil, di-n-butyl phthalate will adsorb moderately to soil and is able to migrate into groundwater under rapid infiltration conditions. It has been suggested that its tendency to form complexes with water-soluble fulvic acids, a component of soils, may aid its transport into groundwater. Di-n-butyl phthalate will also slowly biodegrade, and volatilization from soil is not expected to be a significant loss process. If released to water, di-n-butyl phthalate will adsorb moderately to sediments, and will biodegrade rapidly. A small amount of volatilization will occur, and photooxidation and hydrolysis is not expected to be significant (HSDB 1997).

Di-n-butyl phthalate is metabolized in a very similar fashion to the phthalates described previously, with the monobutyl phthalate being the primary metabolite. Additional metabolites which have been detected are phthalic acid, the omega and omega-1 oxidation products of monobutyl phthalate, and other unidentified polar metabolites, probably conjugates. The toxic mechanism of di-n-butyl phthalate is said to be via mitochondrial uncoupling, which is probably due to an increase in membrane permeability to hydrogen ions and other small ions. It has also been suggested that the monoester of a metabolite of dibutyl phthalate may act as a chelating agent by removing zinc from the testes, and that the resulting testicular zinc deficiency may lead to the observed testicular atrophy in many toxicity studies (HSDB 1997).

A variety of aquatic invertebrates have been tested for di-n-butyl phthalate toxicity. In the scud, *Gammarus fasciatus*, the 1500-hour LC50 was reported to be 0.21 mg/L. The 24-hour LC50 for brine shrimp (*Artemia*) was 8 mg/L, and the 48-hour LC50 for midge larvae, *Chironomus plumosus*, was 0.76 mg/L. Di-n-butyl phthalate was also shown to reduce the number of young produced by *Daphnia magna* at a concentration of 1.8 mg/L. This concentration was also found to decrease the survival of fathead minnow embryos and larvae. An increased incidence of skeletal anomalies were observed in offspring of cyprinodontiform fish, *Rivulus marmoratus*, exposed to 1 mg/L of di-n-butyl phthalate. Di-n-butyl phthalate is not expected to bioaccumulate in fish because it is rapidly metabolized. The log BCF in American oyster, Brown shrimp and sheepshead minnow were 1.5, 1.22, and 1.07, respectively. In fish, log BCFs were also determined to be 3.15 and 3.83 using C-14 labeling. However, these BCFs are not believed to be accurate because di-n-butyl phthalate is rapidly metabolized in fish within four hours, and thus the metabolites would have been included in the measurement of the parent compound for the BCF calculation. In a study using clams, *Neanthes virens*, the measured bioconcentration factors from sediment at two different sites in Portland harbor were 0.59 - 1.1 and 0.14 - 0.25 (HSDB 1997).

The acute oral LD50 for di-n-butyl phthalate is 8,000 - 10,000 mg/kg in rats and 9,000 mg/kg in mice. Other chronic effects that have been observed as a result of oral exposure to di-n-butyl phthalate are reduced weight gain and food consumption, decreased serum cholesterol, lung edema, and increased liver weights. Di-n-butyl phthalate has caused testicular and seminiferous tubular atrophy in males. It has also been shown to result in fetal loss as well as a variety of developmental effects, including neural tube defects, growth retardation, delayed ossification, absence of tail, anophthalmia, twisted legs, hematomas, elongated and fused ribs, absence of tail bones, abnormal or incomplete skull bones, incomplete or missing leg bones and reduced weight of fetuses (HSDB 1997).

Little or no toxicity information was available for birds, but it was shown in one study using Mallard ducks that five months of continuous dietary exposure to di-n-butyl phthalate in food did not result in significant bioaccumulation (HSDB 1997).

E.2.1.6   Di-n-octyl phthalate

Di-n-octyl phthalate is used as a plasticizer, a dye carrier, and for film, wire, cables, and adhesives. If released into soil, di-n-octyl phthalate will sorb strongly to soil and thus is not expected to migrate rapidly into groundwater. However, it has been found in drinking water for which the source was groundwater. If released into surface water, di-n-octyl phthalate will adsorb to seimdent particles, thus providing a transport mechanism for this chemical. Di-n-octyl phthalate will biodegrade slowly after acclimation. No experimental data is available on its ability to photodegrade, but one study estimated its photolysis half-life to be 144 days. Chemical hydrolysis and volatilization are not expected to be significant loss processes for this chemical (HSDB 1997).

Some reports indicate that di-n-octyl phthalate is metabolized to its corresponding monoester derivative, which is then partly excreted unchanged and partly hydroxylated, similar to a variety of other phthalates, described above. However, another source indicates that very little mono-octyl phthalate is produced. Instead, it is reported that the metabolites are mainly a mixture of dicarboxylic acids formed by oxidative scission of the n-octyl side-chain of the mono-octyl phthalate, which are possibly formed by alpha and beta oxidation of the inital omega and (omega-1)-hydroxylation products (HSDB 1997).

Very little information is available on the toxicity of di-n-octyl phthalate in aquatic invertebrates. In one study, a concentration of 1.0 mg/L of di-n-octyl phthalate caused a significant decrease in reproduction of *Daphnia magna*, while 0.32 mg/L had no significant effect. The acute toxicity of di-n-octyl phthalate to fish varies greatly with species. The 7-8 day LC50s for redear sunfish, channel catfish, and largemouth bass were 0.006 mg/L, 0.69 mg/L, and 32.9 mg/L, respectively. Hatching of fathead minnow embryos was decreased at a concentration of 10 mg/L, but was not affected at a concentration of 3.2 mg/L. In an ecosystem model containing phytoplankton, zooplankton, green filamentous algae, snails, mosquito larvae, and mosquito fish, ecological magnification values obtained after three days were 660 for algae, 9,426 for *Daphnia*, 1.16 for fish, 5,300 for mosquitos and 438 for snails, and after 33 days, these values were 28,500 for algae, 2,600 for *Dapnia*, 9,400 for fish and mosquitos, and 13,600 for snails. The higher magnification values after 33 days versus three days might be in part explained by the fact that a final water concentration in the 33 day study was used in determining the bioconcentration values, and this water concentration was low due to the biodegradation of the compound. Nonetheless, this data suggests that di-n-octyl phthalate bioconcentrates in algae and other aquatic organisms (HSDB 1997).

In mammals, di-n-octyl phthalate exhibits very low acute toxicity. The acute oral LD50 for the mouse is 13,000 mg/kg, and for the rat is 30,000 mg/kg. The 12-week LD50 was 3.09 ml/kg in the mouse. Di-n-octyl phthalate has been shown to produce deleterious effects on the developing embryo and/or fetus of the rat. It has also resulted in increased liver weights, and decreased zinc concentrations in the testes of rats. In addition, diets containing di-n-octyl phthalate have resulted in the accumulation of large droplets of fat around the central veins, leading to mild centrilobular necrosis and slight induction of 1 peroxisomal enzyme. Di-n-octyl phthalate has also been found to affect the immune system of the rat. At dose levels that have been shown to cause increased liver weights in mice, no effect on reproduction was observed in a continuous breeding study (HSDB 1997).

Very little information is available on the toxicity of di-n-octyl phthalate to birds. In one study, di-n-octyl phthalate had no adverse effects on 3-day old embryos of the white leghorn chicken (HSDB 1997).

E.2.2   Benzene Derivatives

E.2.2.1   Acetophenone

Acetophenone is released to the environment from a variety of combustion processes and may be released during its manufacture and the manufacture of propylene oxide, kraft bleaching and its use in certain perfumes. In soils, microbial degradation is likely to be the major degradation pathway. It is expected to be moderately to highly mobile in soil and it therefore has the potential to migrate into the groundwater. Evaporation from dry soil surfaces is another important terrestrial fate process. In aquatic systems biodegradation and volatilization are expected to be the major loss processes. Acetophenone is very soluble in water (6,130 mg/L). The estimated biodegradation half-lives in groundwater, river water and lake water samples were 32 days, 8 days and 4.5 days, respectively. Hydrolysis, oxidation, bioconcentration, and adsorption to sediments and suspended particles are not likely to be important fate processes. Oxidation by hydroxyl radicals in air has an estimated half-life of 2.2 days. Other oxidants (eg, ozone) and photolysis do not appear to be important loss mechanism of this compound in air (HSDB 1997).

No field or laboratory bioconcentration/bioaccumulation was available for acetophenone in the literature. A log octanol/water partition coefficient (Kow) of 1.58 and a water solubility of 6,130 mg/L at 25° C was reported for this compound. Based on modeling using these values, the bioconcentration factor is in the range of 5 to 9. Based on these predicted bioconcentration factors, bioconcentration is not expected to be significant in aquatic organisms (HSDB 1997).

No compound-specific toxic mechanisms were available in the literature for this compound. Acetophenone is biodegraded into ethylphenylcarbinol and 1-phenylethanol in rabbits. In rats acetophenone appears to be precursor of mandelic acid, benzoylformic acid, and benzoic acid (HSDB 1997).

The effects on growth, hematological values or macroscopic tissue changes were monitored in groups of 10 male and 10 female rats exposed to 0, 1,000, 2,500 and 10,000 mg/kg acetophenone in the diet for 17 weeks. No effects were observed at any exposure concentration. Some loss of the compound from the feed due to volatilization was reported; therefore, the dietary concentration of 10,000 mg/kg was multiplied by a factor of 0.845 (based on data provided by the investigators) yielding a NOAEL of 423 mg/kg BW/day assuming that a rat consumes a daily amount of food equivalent to 5% of its body weight/day as food (IRIS 1997). The oral LD50 for rats ranged between 900 and 3,200 mg/kg (RTECS 1997).

For aquatic systems, data was only available for the fathead minnow (*Pimephales promelas*) in a flow-through bioassay. A 96-hour LC50 of 162 mg/L and a 96-hour EC50 (loss of equilibrium) of 162 mg/L was reported (HSDB 1997).

No compound-specific information could be found on the toxic effects of acetophenone on birds.

E.2.2.2 Benzidine

Historically benzidine was released into the environment during its production through fugitive emissions and wastewater. Currently benzidine is only produced in the United States for captive consumption with strict regulations that it be maintained in isolated or closed systems which would limit its release to the environment. Benzidine is also used in the manufacture of azo dyes or may be formed during the degradation of benzidine-based dyes. In terrestrial systems, benzidine tends to adsorb to soils (especially acidic soils), form complexes with clay particles, and may be oxidized by metal cations. Degradation rates in soils of 79 percent in 4 weeks have been reported in the literature. In the water column, benzidine will adsorb to suspended clay particles, will be oxidized by naturally occurring metal cations such as $Fe(III)$, and will be lost by reaction with radicals and photolysis. Volatilization from water is not a significant transport process. Its half-life in water is approximately 1 day. This compound will only moderately adsorb to sediments (HSDB 1997).

Benzidine has been reported to bioconcentrate to moderate levels in aquatic systems. In a 42-day flow-through test, bluegills (*Lepomis macrochirus*) were exposed to 14C-benzidine. A bioconcentration factor of 40 was calculated for the edible portions of the fish. Using model ecosystems after a 3-day exposure, bioconcentration factors for fish, mosquitos, snail, and algae were 55, 460, 650, and 2,500, respectively (HSDB 1997).

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Radiolabeled benzidine administered intravenously to rats, dogs, and monkeys was rapidly transferred to the excretory organs (liver, gastrointestinal tract, kidney, and bladder). Significant amounts were also transferred to the lungs. Major routes of excretion in experimental animals given intravenous benzidine are urine and feces. Rhesus monkeys fed azo-dyes, a derivative of benzidine, metabolized a substantial portion of the dyes to free benzidine. Benzidine is rapidly oxidized by a peroxidase/hydrogen peroxide system to products which bind irreversibly to deoxyribonucleic acid (DNA), thereby inhibiting DNA synthesis. Benzidine also induces unscheduled DNA synthesis in Hela cells (HSDB 1997).

Dietary exposure to benzidine at levels of 300 mg/kg for 56 weeks resulted in hepatotoxicity. Necrosis, fatty dystrophy, and hyperplasia were also noted. Exposure of sheepshead minnow (*Cyprinidon variegatus*) to 50 mg/kg of benzidine resulted in tubed heart syndrome with distended pericardia, poor circulation, sparse distribution of melanophores, inability to hatch, abnormal head morphology, scoliosis, and faint red blood cell pigmentation. A 96-hour LC50 OF >20 mg/L was reported for the aquatic macroinvertebrate, scub (*Gammarus pseudolimnaeus*), although the specific conditions of this test were not specified. The 96-hour LC50s for fish range from 4.35 mg/L in lake trout (*Salvelinus namaycush*) to >20 mg/L in fathead minnows (*Pimephales promelas*) (HSDB 1997).

In rats benzidine administered orally at a concentration of 200 mg inhibited testicular DNA synthesis. Covalent binding of benzidine and some of its congeners to hemoglobin was also reported to occur in rats after oral administration (HSDB 1997).

Male and female mice were exposed to benzidine dihydrochloride in the drinking water at 0 to 160 ppm (0 to 27.2 mg/kg/day) for 33 months. Dose-related decreases in body weight gain and survival at all levels were reported; most deaths were caused by tumors. Treatment-related effects included increased incidences of liver cell alterations in females at greater than or equal to 3.8 mg/kg/day, bile duct hyperplasia in females at greater than or equal to 8.2 mg/kg/day and in males at 30.4 mg/kg/day, megakaryocytosis of bone marrow in females at greater than or equal to 11.5 mg/kg/day and in males at greater than or equal to 22.8 mg/kg/day, bladder epithelial hyperplasia in males at 30.4 mg/kg/day, atrophy of the ovary in females at greater than or equal to 15.2 mg/kg/day, brain vacuolization in females at greater than or equal to 3.8 mg/kg/day and males at greater than or equal to 5.7 mg/kg/day, and hemosiderosis of the spleen at greater than or equal to 22.8 mg/kg/day (IRIS 1997).

Benzidine has been reported to be teratogenic to chicks when injected into eggs. No additional compound-specific information could be found on the toxic effects of benzidine to birds.

E.2.2.3   Benzoic Acid

Benzoic acid is typically released into the environment in wastewater or emissions during its production and use as a chemical intermediate and additive. Benzoic acid may also formed by combustion processes and is typically detected in automobile exhaust, refuse combustion, and tobacco smoke. Benzoic acid is commonly added to food products as preservatives and as antimicrobial agents. Benzoic acid is also naturally occurring in the environment (e.g., berries, cranberries, prunes, cloves, bark of wild black cherry tree, scent glands of beavers, and oil of anise seeds). In terrestrial systems, benzoic acid will readily leach into the groundwater due to its low soil adsorption. It biodegrades readily in soil, with a half-life of less than one week. Releases to water also result in rapid biodegradation. Adsorption to sediment and volatilization is not considered significant. Biodegradation is reported to occur

under both aerobic and anaerobic conditions (HSDB 1997).

Bioconcentration factors were highly variable, ranging from 21 in mosquito fish to 2,800 in snails. Bioconcentration in some invertebrate species such as *Daphnia* and snails may be considerable; with BCFs of 1,800 and 2,800, respectively. Bioconcentration in mosquito fish, algae, and mosquito larvae were considerably lower; with BCFs of 21, 100, and 138, respectively (HSDB 1997).

Benzoic acid is rapidly absorbed from the gastrointestinal tract when taken by mouth. It is conjugated with glycine in the liver to form hippuric acid which is rapidly excreted in the urine. With the exception of fowl, virtually all vertebrates excrete benzoic acid as hippuric acid (HSDB 1997).

Very limited toxicity data was available in the literature for biota. Benzoic acid (2%) as found in some preserved pet foods was toxic to cats (HSDB 1997). An LD50 of 1,700 and 1,940 mg/kg was reported in rats and mice, respectively. Toxic effects in mice included somnolence (general depressed activity) and respiratory depression (RTECS 1997).

Benzoic acid at a concentration of 0.5 mg/l did not affect growth of blue-green alga, (*Anabaena* sp.) (RTECS 1997).

No compound-specific information could be found on the toxic effects of benzidine to birds.

E.2.2.4 1,2,4-Trichlorobenzene

1,2,4-trichlorobenzene enters the environment through its manufacture and its major use as a dye carrier. Intermediate uses for this compound involve the manufacture of herbicides and higher chlorinated benzenes, dielectric fluid, solvent, heat-transfer medium, septic tank and drain cleaners, wood preservatives, and abrasive formulations. 1,2,4-trichlorobenzene is often used as a replacement for polychlorinated biphenyls in the electrical industry. It is also formed during the combustion of chlorine-containing polymers and through the dechlorination of hexachlorobenzene by anaerobic sewage sludge. It is a synthetic organic chemical and is not known to occur as a natural compound. It adsorbs readily to soil and, therefore, is not expected to leach to an appreciable degree into the groundwater. Experimental data has reported biodegradation to occur in soil. In surface water, it may adsorb to sediment and evaporate. It does not readily hydrolyze in surface water, but may undergo slow biodegradation (HSDB 1997).

The bioconcentration factor for 1,2,4-trichlorobenzene by rainbow trout (*Salmo Gairdneri*) exposed over 119 days was studied in a flow-through system. Fish exposed to water containing 3.2 ng/L had a BCF of 1,300 while fish exposed to a concentration of 52 ng/L had a BCF of 3,200. Certain developmental stages of rainbow trout (*Salmo gairdneri*) accumulated more 1,2,4-trichlorobenzene than other stages. The bioconcentration factor was approximately 10 times as great at hatching as in alevins. These differences can be reduced, but not eliminated, by expressing the data on the basis of lipid weight. Daphnids (*Daphnia* sp.) continuously exposed to a mean measured aqueous concentration of 3.1 ug/L of 1,2,4-trichlorobenzene had an equilibrium BCF of 142 (HSDB 1997).

Contaminated food was prepared by exposing pink shrimp (*Penaeus duorarum*) to 10 ug/L 1,2,4-trichlorobenzene-UL-(14)C for 12 days; the mean whole body concentration of trichlorobenzene in the exposed shrimp was 0.59 mg/kg. Juvenile spot (*Leiostomus xanthurus*) were fed the trichlorobenzene contaminated shrimp at a daily ration of 10

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percent body weight for 28 days; they accumulated less than 0.05 mg/kg trichlorobenzene (detection limits). Spot which were exposed to 10 ug/L trichlorobenzene in water for 28 days and fed uncontaminated food exhibited a BCF of approximately 100. Spot, exposed simultaneously to contaminated food and the same water concentration also exhibited a BCF of 100. Results were compared with data from a trichlorobenzene bioaccumulation study with freshwater species; both studies indicated that trichlorobenzene was accumulated moderately from contaminated water and accumulation from contaminated food was negligible (HSDB 1997).

Specific toxic mechanisms for this compound were not available in the literature. In general, the halogenated benzenes appear to increase the activity of microsomal reduced nicotinamide adenine dinucleotide cytochrome p450 dependent enzyme systems. 1,2,4-Trichlorobenzene is absorbed from the GI tract, intact skin, and lung. In rats 1,2,4-trichlorobenzene was dehalogenated by hepatic micosomes. Seven days after treatment, the excretion of radioactivity into the urine and feces was about 66 and 17 percent of the given dose, respectively. 1,2,4-Trichlorobenzene and its dehalogenated derivatives in the breath represented about 2.1% of the dose; the excretion of radioactivity reached a plateau in 3 days. Biliary and fecal excretion amounted to 45.3 and 20% of the dose, respectively. High initial levels were recorded in body fat 12 and 24 hr after treatment; on days 3 and 7 the body fat levels were still slightly higher than those in other organs (HSDB 1997).

A multigenerational reproductive study was used to determine the chronic toxicity values of 1,2,4-trichlorobenzene in rats. At birth the F0 generation litters (17-23 litters/dose group) were randomly reduced to 4 males and 4 females. Male and female progeny were dosed with 0, 25, 100 or 400 mg/kg of 1,2,4-trichlorobenzene in the drinking water. During the study maternal weights, litter size, neonate sex and weight, and 24-hour food and water intake were recorded. Blood samples and organs were collected on days 27 and 95 of age from selected rats from each group for chemistry determinations (i.e., glucose, BUN, creatinine, Na, K, Cl, uric acid, Ca, P, cholesterol, triglyceride, bilirubin, alkaline phosphatase, SGOT, SGPT, LDH, CPK, protein, globulin and albumin), and organ weights (i.e., liver, kidney, uterus, adrenals, lungs, heart and gonads). Similar procedures were performed with the F1 generation. The study ended when the F2 generation was 32 days old. Fertility (as indexed by conception rate of dams) of the F0 and F1 generation rats was not affected by treatment. A LOAEL was derived from a significant increase (11percent in males, 13percent in females) in adrenal gland weights observed in the 400-mg/kg groups of males and females of the F0 and F1 generations. The NOAEL was determined to be 100 mg/kg from the mid-dose group. The LOAEL was determined to be 400 mg/kg on the basis of increased adrenal gland weight (IRIS 1997).

Five rats/group were dosed with 53 mg/kg/day of 1,2,4-trichlorobenzene in corn oil by gavage. Microscopic examination of the 1,2,4-trichlorobenzene-treated rats showed moderate vacuolization of the zona fasciculata; the control group showed only slight vacuolization. Twenty-four hour urine and serum specimens were collected prior to post mortem examination. A 14 percent increase in absolute adrenal gland weight was observed and a 13 percent adrenal gland/body weight ratio was observed. In addition, the treated rats had decreased serum corticosterone levels when compared with controls (IRIS 1997).

The maternal reproductive and hepatic effects of 1,2,4-trichlorobenzene on rats that were dosed with 0, 36, 120, 360 or 1,200 mg/kg/day of 1,2,4-trichlorobenzene on days 9 through 13 of gestation. Among the treatment groups of 9 dams/group, alteration of embryonic parameters was noted only in the 360 mg/kg/day group (all of the dams in the 1200

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mg/kg/day group died). The observed changes included significant retardation of all four growth criteria (i.e., head length, crown-rump length, somite number, and protein content). 1,2,4-trichlorobenzene did not cause increased resorptions, embryolethality or teratogenicity. This study also demonstrated significantly increased xenobiotic hepatic enzyme activity at 120 and 360 mg/kg/day (IRIS 1997).

Oral doses of 1,2,4-trichlorobenzene (173.6 mg/kg) to Rhesus monkeys resulted in severe weight loss and tremors followed by death. Clinical and biochemical changes were reversible if the doses were discontinued. Oral doses to rats indicate that this compound may be an enzyme inducer of the phenobarbitol type. Generally, halogenated benzenes appear to increase the activity of systems that enhance the metabolism of a wide variety of xenobiotics. It is readily absorbed by the gastrointestinal tract, skin, and lung (HSDB 1997).

Oral LD50s for rats and mice were 756 mg/kg and 300 mg/kg, respectively. The toxic effects observed in rats included somnolence (general depressed activity) and convulsions or effect on seizure threshold. The toxic effects observed in mice included, altered sleep time (including change in righting reflex), convulsions or effect on seizure threshold, and ataxia (RTECS 1997).

Macrobenthic animal communities that colonized sand-filled aquaria were exposed to 1,2,4-trichlorobenzene. In one test, communities established by planktonic larvae entrained in continuously supplied unfiltered seawater for 50 days were exposed to waterborne 1,2,4-trichlorobenzene for 6 days; in the second test, the toxicant was added to the sediment before 8 weeks of colonization. The concentration that affected community structure were usually two orders of magnitude lower for waterborne 1,2,4-trichlorobenzene than for sediment-bound 1,2,4-trichlorobenzene, but the same types of organisms were affected by each route of exposure. The lowest 1,2,4-trichlorobenzene concentration (measured) that affected average numbers of individuals exposed via the water were 0.04 mg/l for mollusks, 0.4 mg/L for arthropods, and 4 mg/L for annelids. Average number of species was significantly lower than the control at 4 mg/L. For 1,2,4-trichlorobenzene exposures via the sediment, the lowest concentration that affected average numbers of individuals were 100 mg/kg for mollusks and echinoderms, and 1,000 mg/kg for arthropods and annelids. The average number of species in experimental aquaria was significantly lower than the control at greater than or equal to 100 mg/kg. 1,2,4-Trichlorobenzene persisted in sediments, but some leached into water throughout the 8 week exposure via sediment (HSDB 1997).

The 96-hour LC50 for fathead minnows (*Pimephales promelas*), bluegill (*Lepomis macrochirus*) and sheepshead minnows (*Cyprinodon variegatus*) were 2.9, 3.4, and 21.4 mg/L, respectively (HSDB 1997).

The porphyrinogenic action of 1,2,4-trichlorobenzene was examined in 17 day old embryos, one-day old chicks, 18 day old chickens and adult Japanese quail. The quail was the most sensitive species towards 1,2,4-trichlorobenzene induced porphyria. The chick embryo was non-responsive to this compound. The liver porphyrins of Japanese quail were increased in a dose-dependent manner 1 day after 1,2,4-trichlorobenzene treatment. Elevation in porphyrin levels in quail was associated with a comparable increase in delta-aminolevulinic acid synthetase (ALA-S) activity 1 day after 1,2,4-trichlorobenzene treatment. Multiple administration of 1,2,4-trichlorobenzene produced only a slight increase in liver porphyrin levels and delta-aminolevulinic acid synthetase activity in quail. There was a marked induction in ferrochelatase activity suggesting increased porphyrin turnover. Liver glutathione and glutathione S-tranferase activity were significantly increased (HSDB 1997).

White Leghorn chick embryos and day old chicks were used to study the effects of monochlorobenzene, para-dichlorobenzene, and 1,2,4-trichlorobenzene on liver porphyrin levels and the effects of 1,2,4-trichlorobenzene on hepatic drug metabolism. The chlorobenzenes were administered to fertilized eggs at doses of 10 and 40 mg/egg, and the embryos were sacrificed at 24 hours post-treatment. The chlorobenzenes were administered orally to day old chicks at doses of 200 and 800 mg/kg body weight. The chlorobenzenes at the 40 mg level had no effect on the porphyrin contents of chick embryo liver, but increased hepatic prophyrin levels in day old chicks at a dose of 800 mg/kg were observed. The order of effectiveness from greatest to least was 1,2,4-trichlorobenzene, para-dichlorobenzene, and monochlorobenzene. Treatment of day old chicks with 800 mg/kg chlorobenzenes also increased the porphyrin content of the bile with a similar order of effectiveness. Treatment with any of the chlorobenzenes had no significant effect on cytochrome p450, cytochrome-b5, glutathione-S-transferase, 7-ethoxyresorufin-O-dethylase, or 7-ethoxycoumarin-O-deethylase in chick embryos or biliary excretion of porphyrin. The data indicated that the porphyrinogenic actions of monochlorobenzene, para-dichlorobenzene and 1,2,4-trichlorobenzene and their capacity to induce microsomal monooxygenases depend on the stage of development of chicks as well as the degree of chlorination of the chemical (HSDB 1997).

E.2.2.5   Bis (2-Chloroisopropyl) Ether

Bis (2-chloroisopropyl) ether is no longer commercially produced in the United States. It was previously used as a solvent and as an extractant. Bis(2-chloroisopropyl) ether may form as a by-product of propylene oxide production. It has also been found in industrial waste water and in natural water (HSDB 1997).

Bis (2-chloroisopropyl) ether hydrolyzes rapidly in water (half-life of <38.4 seconds) or moist soil, therefore, biodegradation, and adsorption to soil and sediment are not expected to be significant fate processes (HSDB 1997).

No data was available in the literature regarding BCFs for this compound, although due to its rapid hydrolysis rates, bioconcentration is not considered to be an important rate process (HSDB 1997).

No information about toxic mechanisms of bis(2-chloroisopropyl) ether were available in the literature. Male rats were given single oral doses of (14)carbon labeled bis(2-chloroisopropyl) ether at 90 mg/kg. Metabolites identified in urine were 2-(2-chloro-1-methylethoxy)propanoic acid at approximately 36 percent and N-acetyl-s-(2-hydroxypropyl)-l-cysteine at approximately 19 percent (HSDB 1997).

Bis-(2-chloroisopropyl) ether (98.5 percent pure) was incorporated into the diets of 102 male and female mice at levels of 0, 80, 400, 2,000, or 10,000 mg/kg for up to 104 weeks. Comprehensive hematological, blood biochemical, and urinalysis determinations were performed on 7 mice per sex per group at 13, 26, and 52 weeks; on 6 mice per sex per group at 78 weeks; and on the remaining mice at 104 weeks. Hemosiderin deposition in the spleen was seen in the males on the 10,000 mg/kg diet and in females receiving both the 2,000 and 10,000 mg/kg diet. Decreases in hemoglobin concentration and red blood cell counts were also observed. These findings indicate a treatment-related increase in erythrocyte destruction. A NOAEL of 2,000 mg/kg for males and 400 mg/kg for females was indicated. These doses were determined by the investigators to be equivalent to 198 and 35.8 mg/kg/day for males and females, respectively (IRIS 1997).

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No compound-specific information could be found on the toxic effects of bis-(2-chloroisopropyl) ether to aquatic organisms or birds.

E.2.2.6   2,6-Dinitrotoluene

No specific use information is available for 2,6-dinitrotuolene, however, dinitrotoluenes in general are used in organic synthesis, dyes, and explosives. It is expected to biodegrade if released to the soil. Experiments with 2,6-dinitrotoluene in sandy loam and sandy silt loam indicates that this compound is mobile. It is also expected to biodegrade in water. Photooxidation is anticipated to occur in surface water and is not expected to adsorb to sediments or suspended solids, or volatilize. A bioconcentration factor of 5,225 was measured for an algal biomass in a model waste stabilization pond, however, an estimated bioconcentration factor of 12 is recommended based on an estimated partition coefficient and regression equations (HSDB 1997).

The primary subacute toxic effects are seen in red blood cells, the nervous system, and the testes. Male mammals fed 2,6-dinitrotoluene exhibited decreased spermatogenesis, aspermatogenesis, or testicular atrophy. Non-functioning ovaries were found in female mammals. Higher dietary doses result in death. There is no evidence for preferential uptake or retention of 2,6-dinitrotoluene among body tissues. Urine is the major route of elimination for this compound. 2,6-Dinitrotoluene was found to form covalent bonds with DNA in liver cells with lesser binding to the lung and intestine (HSDB 1997).

E.2.2.7   3,3-Dichlorobenzidine

3,3-Dichlorobenzidine enters the environment as emissions or in wastewater during its production or use as an intermediate in the manufacture of pigments and dyes or as an ingredient in rubber and plastic compounding. In surface water, it adsorbs rapidly to sediment and particulate matter where it becomes tightly bound or, possibly chemically bound. It undergoes rapid photooxidation forming 3-chlorobenzidine and benzidine. When released on land, it binds tightly to soil components. Mineralization in the soil does occur but at a very slow rate. Experimental BCFs have been determined for this compound to be as follows: 495-507 for bluegill sunfish, 610 in Golden ide, and 940 in algae. Therefore, 3,3-dichlorobenzidine is expected to bioconcentrate in aquatic organisms (HSDB 1997).

The acute toxicity of 3,3-dichlorobenzidine is relatively low, with 7-day oral LD50s ranging from 352-488 mg/kg/day for mice. An oral LD50 for the rat was determined to be 3820 mg/kg . However, oral intake of 3,3-dichlorobenzidine has resulted in gastrointestinal congestion and hemorrhaging in rats. Covalent binding of this compound to hepatic lipids has also been reported following oral doses. It has also been classified as a carcinogen in laboratory animals (HSDB 1997).

Information on the toxic effects of 3,3'-dichlorobenzidine to birds was not available at the time of this report.

E.2.2.8   Hexachlorobenzene

Hexachlorobenzene (HCB) is formed as a waste product in the production of several chlorinated hydrocarbons and is a contaminant in some pesticides. Hexachlorobenzene is very persistent in the environment due to its chemical stability and resistance to biodegradation. In water, significant amounts will partition from the water column to the sediment and suspended matter. Volatilization from water is rapid, however, its strong

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adsorption to sediment precludes its rapid disappearance from aquatic systems. Hexachlorobenzene strongly adsorbs to soil and will generally not leach into the ground water. This compound will bioconcentrate in fish and transfer through trophic levels. Some log bioconcentration factors are as follows: trout (3.7-4.3); sunfish (3.1-4.3); and fathead minnow (4.2-4.5) (Howard 1989).

Dietary hexachlorobenzene induced experimental porphyria and partial uncoupling of oxidative phosphorylation of liver mitochondria in rats. This uncoupling is due to the action of pentachlorophenol, which was formed by metabolism of the hexachlorobenzene (Masini et al. 1985).

E.2.2.9   4-Methylphenol

4-Methylphenol, also commonly referred to as p-cresol, is released in auto and diesel exhaust, during coal tar refining, wood pulping, and during its use in manufacturing and metal refining. It is also used as a gelatinizing and waterproofing agent in explosives and in the synthesis of TNT, urethane polymers, flexible and rigid foams, surface coatings, and dyes (HSDB 1997).

When released into water, biodegradation is the dominant loss mechanism. Volatilization and adsorption to sediment are not considered important fate processes. Photolysis is expected only in the surface waters of oligotrophic lakes. Its fate in soil has not been extensively studied. However, it is anticipated to biodegrade in soil. Given an estimated BCF of 12, 4-methylphenol is not expected to bioconcentrate appreciably (HSDB 1997).

In an outdoor experimental stream, this compound's primary effect on the flora and fauna community was the interference with the photosynthetic and respiration processes (HSDB 1997).

In mammals, signs of acute 4-methylphenol poisoning include muscular convulsions, coma, and death from respiratory paralysis. The acute oral LD50 for 4- methylphenol in dogs is 4 mg/kg. 4-Methylphenol is a general protoplasmic poison and is toxic to all cells. It is also a potent hepatocarcinogen (HSDB 1997).

No information on the toxic effects of 4-methylphenol in birds was available at the time of this report.

E.2.2.10   4-Nitroaniline

4-Nitroaniline may be released to the environment from process and waste emissions involved in its production or use as a chemical intermediate and through stack emissions from hazardous waste incineration. It is also used as a veterinary pharmacological agent and as a chemical intermediate for the production of antioxidants, dyes, pigments, and gasoline gum inhibitors (HSDB 1997).

In soil, this compound undergoes covalent chemical bonding with humic materials which can result in its chemical alteration to a latent form and prevent leaching. However, experimental data has shown 4-nitroaniline to have high to very high mobility in soil. Photodegradation may occur on soil surfaces that are exposed to sunlight. It is not expected to volatilize from moist or dry soils. In water, it will covalently bond to humic materials in the water column and sediment. Photodegradation in water may be possible. Aquatic

hydrolysis, volatilization, and bioconcentration are not considered significant, and this compound is generally resistant to biodegradation. In a 6-week bioconcentration test, experimental BCFs ranged from 2.9-3.6 at a concentration of 0.5 mg/L and ≤ 10 at a concentration of 0.05 mg/L. It was also found to have no or low bioaccumulation potential in carp, and a BCF of 4.4 in zebrafish at a concentration of 0.21 umol/L. Therefore, bioconcentration is not expected to be an important fate process for 4-nitroaniline (HSDB 1997).

4-nitroaniline has been shown to be hematoxic, causing methemoglobinemia, Heinz body formation and a physiological, compensatory reaction to maintain erythrocyte mass. In addition, histopathological changes in the spleen, liver, and bone have been observed in mice which had been administered 4-nitroaniline in corn oil via gavage. Oral doses of 4-nitroaniline to pregnant mice and rats have resulted in decreased numbers of viable litters and reduced pup viability, but only at doses which caused maternal toxicity (HSDB 1997).

No information on the toxicity of 4-nitroaniline to birds was available at the time of this report.

E.2.2.11   Dibenzofuran

Dibenzofuran is a component of coal tar and is released to the environment through its manufacture and use. It is also released during coal, biomass, refuse, diesel fuel, residual oil, and tobacco combustion. It is also a component of heat-transfer oils, a carrier for dyeing and printing textiles, an intermediate for production of dyes, antioxidants, and plastics (HSDB 1997).

With a log octanol-water coefficient (log Kow) of 4.12, dibenzofuran is expected to have very low to no mobility in soil. In water, it is expected to adsorb strongly to sediment and particulate matter in the water column. Based on its vapor pressure and water solubility, it is expected that dibenzofuran may volatilize from water. Biodegradation occurs in soil and water if sufficient microbial populations are present, otherwise, microbial degradation is slow. Biodegradation is also slow when oxygen is limited (HSDB 1997).

Experimental BCFs were determined for dibenzofuran in a 33-day model ecosytem study. The following BCFs were calculated: 82 for algae, 2858 for snail, 2094 for mosquito, and 947 for fish. In a 28-day flow-through study using fathead minnow, BCFs ranged from 1100 to 1700. However, over 97% of the dibenzofuran was eliminated in 2 days during depuration tests. Experimental BCFs of 850-2200 were also obtained from an 8-week test using guppies. Given these experimental BCFs, dibenzofuran would be expected to bioconcentrate in aquatic organisms. However, its rapid elimination from the body would reduce this potential (HSDB 1997).

Information on the toxicity of dibenzofuran could not be located at the time of this report.

E.2.3   Polycyclic Aromatic Hydrocarbons (PAHs)

Polycyclic aromatic hydrocarbons (PAHs) consist of hydrogen and carbon in the form of two or more fused benzene rings. There are numerous PAH compounds, each differing in the number and arrangement of benzene rings. The low molecular weight PAHs (2 -3 rings) tend to be more acutely toxic, while higher molecular weight compounds (4 - 7 rings) tend to be carcinogenic, teratogenic, or mutagenic (See Table E.2.3.A) (Eisler 1987b).

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Chemical Properties of PAHs Identified as COPCs for the Ecological Risk Assessment

Compound	Benzene Rings	Chemical Formula	Molecular Weight	Octanol/Water Partition Coefficient (Log Kow)	Solubility in Water at 20 to 25° C (mg/L)
2-methylnaphthalene	2	$C_{11}H_{10}$	142.20	3.86	NA
acenaphthene	3	$C_{12}H_{10}$	154.21	3.92	Insoluble
acenaphthylene	3	$C_{12}H_8$	152.20	4.07	16.1
anthracene	3	$C_{14}H_{10}$	178.22	4.45	1.29
fluorene	3	$C_{13}H_{10}$	166.21	NA	Insoluble
naphthalene	2	$C_{10}H_8$	128.16	3.30	30
phenanthrene	3	$C_{14}H_{10}$	178.22	4.57	0.6
benzo(a)anthracene	4	$C_{18}H_{12}$	228.28	5.61	0.014
benzo(a)pyrene	5	$C_{20}H_{12}$	252.30	6.04	0.0038
benzo(b)fluoranthrene	5	$C_{20}H_{12}$	252.30	6.12	0.0012
benzo(g,h,i)perylene	6	$C_{22}H_{12}$	276.34	6.58	0.00026
benzo(k)fluoranthene	5	$C_{20}H_{12}$	252.32	6.84	0.00076
chrysene	4	$C_{18}H_{12}$	228.28	5.61 to 5.91	0.0020
dibenz(a,h)anthracene	5	$C_{22}H_{14}$	278.33	6.50	0.0005
fluoranthene	4	$C_{16}H_{10}$	202.26	4.9	Insoluble
indeno(1,2,3-c,d)pyrene	6	$C_{22}H_{12}$	276.34	6.584	0.062
pyrene	4	$C_{16}H_{10}$	202.24	4.88	0.135

In general, it appears that toxicity associated with PAHs is due not to the initial compound, but with metabolized intermediates. The majority of the enzymatic activity associated with the metabolism of PAH compounds takes place in the liver (Fourman 1989). The first step in the metabolic process is the oxidation of PAHs by cytochrome P450 and P448 enzyme systems. The metabolic by-products go through a series of reactions, ultimately forming diol-epoxides and phenol-oxides, which are believed to be the carcinogenic intermediates of PAHs (Stein et al. 1990). These compounds have the ability to form DNA adducts by covalently bonding with genetic material (Varnasi et al. 1989). Metabolic activation of PAHs can also involve the formation of free radicals and carbonium ions as metabolized intermediates; these are potential carcinogens and will affect metabolic pathways (HSDB 1997).

PAHs are also potent immunotoxic compounds, suppressing the humoral and cell-mediated immune response. Many PAHs have been shown to adversely affect host tumoricidal activities, resulting in tumor formation (Peakall 1993). Application of carcinogenic PAHs to skin leads to destruction of

sebaceous glands, hyperplasia, hyperkeratosis, ulceration, and potential tumor induction (Eisler 1987b).

Target organs for PAH toxic effects are diverse because these compounds are extensively distributed in the body and they each tend to selectively attack proliferating cells. Damage to the hematopoietic and lymphoid system in experimental animals is common. Target organs can also be species specific. In rats, the target organs for 7,12-dimethylbenz(a)anthracene are skin, small intestine, kidney and mammary gland, whereas in fish the primary target organ is the liver (Eisler 1987b).

When PAHs are taken up by organisms, there is a wide range of metabolism rates between PAH compounds. For example, benzo(a)pyrene accumulations in bluegill (*Lepomis macrochirus*) were 10 times greater than naphthalene, but benzo(a)pyrene was extensively metabolized, whereas naphthalene was not (Eisler 1987b). Because the more hydrophobic PAHs (e.g., benzo(a)pyrene), show a high affinity for binding to dissolved humic materials and have comparatively rapid biotransformation rates, these interactions may lessen or negate bioaccumulation and food chain transfer of hydrophobic PAHs (Eisler 1987b).

In addition to a wide range of metabolism rates between PAH compounds, there is also a very wide range of metabolism rates of PAH compounds between species. Mammals, fish, and most crustaceans possess microsomal oxidase enzymes necessary for PAH metabolism. Some organisms (e.g., algae, zooplantkon, plants, mussels, scallops, and snails) lack these enzymes and are unable to efficiently metabolize PAHs (Eisler 1987b). These species may tend to bioconcentrate PAHs to many times above the ambient concentrations.

Within a single species, bioconcentration rates tend to vary between PAH compound. In general, bioconcentration factors tend to increase as the molecular weight of the PAH increases, as the octanol/water partition coefficient value increases, as the dissolved organic matter increases, and as lipid content of the organism increases (See Table E.2.3.A). Bioconcentration/bioaccumulation in organisms that are able to metabolize PAHs is considered to be short-term, and is not considered an important fate process (HSDB 1997). Laboratory studies may overestimate accumulation rates compared to the environment. In the environment PAHs tend to strongly and rapidly adsorb to sediments limiting their bioavailability, whereas in laboratory bioconcentration studies the PAHs are in the water column and available to the test organisms. For example, PAHs were analyzed in surficial sediments & benthic organisms collected from southeastern Lake Erie, near a large coal-fired power plant. Sediment concentrations (530-770 micrograms per kilogram [μg/kg] total PAHs) were relatively homogenous throughout most of the 150 square kilometer area, although river and near-shore concentrations reached 4,000 μg/kg. Oligochaete worms did not bioconcentrate any of the PAHs. Midges (Chironomide) collected 1 km offshore exhibited bioconcentration of 5 PAH compounds. Further offshore, midges did not appear to be bioconcentrating PAHs and the concentration in the midges were at near equilibrium with the surrounding sediments (HSDB 1997).

Many studies evaluating the toxicity of PAH compounds were performed using mixtures of PAHs rather than individual PAHs. Below are several relevant studies that evaluated the toxic effects of mixtures of PAHs on aquatic organisms and birds.

The toxic effect of PAHs, (benzene, toluene, naphthalene, 1-methylnaphthalene, anthracene, 9-methylanthracene, phenanthrene) on the productivity of various marine planktonic algae (*Dunaliela biocula, Phaeodactylum tricornutum,* and *Isochysis galbana*), increased with increasing number of aromatic rings (see Table E.2.3.A). The methylated compounds were most toxic. Taxonomic differences in sensitivity between the species of algae evaluated was not demonstrated (HSDB 1997).

Combined field and lab studies were conducted to assess the possible role of contaminated bottom sediments to neoplastic disease in fish from eastern Lake Erie and upper Niagara River. Correlations were observed between PAH levels in the sediment and neoplasms in feral fish, and the induction of neoplasms in bullheads (*Ictalurus nebulosus*) (HSDB 1997).

Buffalo river sediment extracts contained PAH compounds which caused skin darkening, hyperplasia, skin papillomas, mild coarsening and local pigmentations in the brown bullhead (*Ictalurus nebulosus*). Sixteen PAH compounds were identified in the sediment extract: fluorene, phenanthrene, anthracene, fluoranthene, 2-methylphenanthrene, pyrene, 2-methylanthracene, benzanthracene, chrysene, perylene, benzo(f)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, dibenz(a,h)anthracene, benzo(g,h,i)perylene, and indeno(1,2,3-c,d)pyrene (HSDB 1997).

Two studies were available in the literature evaluating the toxic effects of PAHs to birds. No mortality was observed in mallards fed diets containing 4,000 mg/kg of PAHs (consisting primarily of naphthalenes, naphthenes, and phenanthrene) for a period of 7 months. Compared to controls, livers from exposed mallards were 25 percent heavier and had a 30 percent increase in blood flow to the liver (Eisler 1987b).

Embryo toxicity was also evaluated in mallards. Researchers applied various PAH compounds (in a comparatively innocuous synthetic petroleum mixture) externally to the surface of mallard eggs. 7,12-dimethylbenz(a)anthracene was the most embryotoxic compound evaluated. A concentration of 0.002 µg/egg (equivalent to about 0.036 µg/kg fresh weight, based on an average egg weight of 55 grams) caused 26 percent mortality and a number of toxic responses in the surviving birds in the 18 day study. Similar effects were also observed when eggs were exposed to chrysene concentrations at 0.015 µg/egg. Benzo(a)pyrene at a concentration of 0.01 µg/egg caused 60 percent mortality in 18 days (Eisler 1987b).

Below are compound specific toxicity profiles for 2-methylnaphthalene (E.2.4.1), acenaphthene (E.2.4.2), acenapthylene (E.2.4.3), anthracene (E.2.4.4), fluorene (E.2.4.5), naphthalene (E.2.4.6), phenanthrene (E.2.4.7), benzo(a)anthracene (E.2.4.8), benzo(a)pyrene (E.2.4.9), benzo(b)fluoranthrene (E.2.4.10), benzo(g,h,i)perylene (E.2.4.11), benzo(k)fluoranthene (E.2.4.12), chrysene (E.2.4.13), dibenz(a,h)anthracene (E.2.4.14), fluoranthene (E.2.4.15), indeno(1,2,3-cd)pyrene (E.2.4.16), pyrene (E.2.4.17), acetophenone (E.2.4.18).

E.2.3.1   2-methylnaphthalene

2-Methylnaphthalene is a component of crude oil and a product of combustion which is released to the environment during natural fires. Emissions from petroleum refining, coal tar distillation, and gasoline and diesel fueled engines are major contributors of 2-methylnaphthalene to the environment. 2-methylnaphthalene may also be released to the environment via manufacturing effluents and the disposal of waste byproducts. Because of the widespread use of 2-methylnaphthalene in a variety products, 2-methylnaphthalene is also released to the environment through landfills, municipal waste water treatment facilities and waste incinerators (HSDB 1997).

2-methylnaphthalene should biodegrade rapidly in the environment where microorganisms have acclimated to PAHs and at a moderate rate in unacclimated soils and aquatic systems. Based on chemical properties, hydrolysis and bioconcentration of 2-methylnaphthalene should not be important fate processes in the environment; photolysis, adsorption, and volatilization are. The direct photolysis half-life for 2-methylnaphthalene in sunlit waters at midday, midsummer and 40 deg N latitude was predicted to be 54 hours. Photolysis is also likely to occur in air and on sunlit soil surfaces. A measured Koc of 8,500 indicates

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2-methylnaphthalene will be immobile in soil. In aquatic systems, 2-methylnaphthalene may partition from the water column to organic matter contained in sediments and suspended solids. A Henry's Law constant of 5.18 X 10-4 atm-cu m/mole at 25 deg C suggests volatilization of 2-methylnaphthalene from environmental waters may be important. The volatilization half-lives from a model river and model pond (the latter considers the effect of adsorption) have been estimated to be 5.5 hours to 77.7 days, respectively. 2-methylnaphthalene is expected to exist entirely in the vapor phase in ambient air (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. Below is a list of bioaccumulation/bioconcentration rates reported by Eisler (1987b) and HSDB (1997) for this compound:

Taxonomic Group	BCF/BAF	Exposure Period
Clams	8.1	4 hour

No toxic mechanisms other than those described above for PAHs were located in the literature for this compound.

Only one study was located that evaluated the toxicity of 2-methylnaphthalene to mammals. 2-methylnaphthalene administered orally at a concentration of 5 mg/kg was lethal to rats (HSDB 1997).

In the aquatic environment, 2-methylnaphthalene was acutely toxic to invertebrates and fish at concentrations ranging from 1.1 to 5.0 milligrams per liter (mg/L). The 96-hour Median Lethal Concentration (LC50) for grass shrimp was 1.1 mg/L (Neff 1985). The 48-hour and 96-hour LC50 for larval Dungeness crab (*Cancer magister*) was 5.0 and 1.3 mg/L, respectively (HSDB 1997). The 24-hour LC50 for sheepshead minnow was 2.0 mg/L (Neff 1979). No chronic toxicity data for 2-methylnaphthalene was available in the literature.

No compound-specific information could be found on the toxic effects of 2-methylnaphthalene to birds.

E.2.3.2   Acenaphthene

Acenaphthene is a component of crude oil and a product of combustion which is released to the environment during natural fires. Emissions from petroleum refining, coal tar distillation, coal combustion, and diesel fueled engines are major contributors of acenaphthene to the environment. Acenaphthene is also used as a chemical intermediate and may be released to the environment via manufacturing effluents and the disposal of waste byproducts of petrochemical, pesticide, and wood preservative industries. Because of the widespread use of acenaphthene a variety products, acenaphthene is also released to the environment through landfills, municipal waste water treatment facilities, and waste incinerators (HSDB 1997).

Acenaphthene should biodegrade rapidly in the environment. The reported biodegradation half-lives for acenaphthene in aerobic soil and surface waters range from 10 to 60 days and 1 to 25 days, respectively. Acenaphthene is more persistent in the environment in anaerobic conditions or at high concentrations due to toxicity to micro-organisms. The calculated Koc range of 2,065 to 3,230 indicates that acenaphthene will be slightly mobile in soils. In aquatic systems, acenaphthene can partition from the water column to organic matter

contained in sediments and suspended solids. A Henry's Law constant of 1.55×10^{-4} atm-cu m/mole at 25 degrees Celsius (° C) suggests volatilization of 2-methylnaphthalene from environmental waters may be important. The volatilization half-lives from a model river and model pond, the latter considers the effect of adsorption, have been estimated to be 11 hours to 39 days, respectively. Acenaphthene is expected to exist entirely in the vapor phase in ambient air (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. Below is a list of bioaccumulation/bioconcentration rates reported by Eisler (1987b) and HSDB (1997) for this compound:

Taxonomic Group	BCF/BAF	Exposure Period (Exposure Conc.)
Bluegill Sunfish (*L. macrochirus*)	387	28 day
Bluegill Sunfish (*L. macrochirus*)	2.6	28 day (8.94 µg/L)

No toxic mechanisms other than those described above for PAHs were located in the literature for this compound.

Only one study was available evaluating the effects of acenaphthene on mammals. Acenaphthene was orally administered to seven young rats at a concentration of 2,000 mg/kg BW for a period of 32 days. Effects observed at the end of the study included loss of body weight, changes in peripheral blood, increased aminotransferase levels in blood serum, and mild morphological damage to both the liver and kidney. The rats also showed mild bronchitis and localized inflammation of peribronchial tissue (HSDB 1997).

In the aquatic environment, acenaphthene has been shown to be toxic to a variety of organisms. In fish the 96-hour LC50s for brown trout (*Salmo trutta*), fathead minnows (*Pimephales promelas*), bluegills (*Lepomis macrochirus*), channel catfish (*Ictalurus punctatus*), and sheepshead minnows (*Cyprinodon variegatus*) were 0.58, 1.6, 1.7, 1.7, and 2.2 mg/L, respectively. The 96-hour LC50s for invertebrates ranged from 0.97 mg/L in mysid shrimp (*Mysidopsis bahia*) to 2.0 mg/L for snails (*Aplexa hynorum*) (HSDB 1997). No chronic toxicity data for acenaphthene was available in the literature.

No compound-specific information could be found on the toxic effects of acenaphthene on birds.

E.2.3.3   Acenapthylene

Acenaphthylene is a component of crude oil, coal tar and a product of combustion which may also be released to the environment in emissions from petroleum refining and coal tar distillation. Acenaphthylene is contained in a variety of coal tar products and may be released to the environment via manufacturing effluents and the disposal of manufacturing waste byproducts. Because of the widespread use of materials containing acenaphthylene, releases to the environment also may occur through municipal waste water treatment facilities and municipal waste incinerators.

Acenaphthylene should biodegrade in the environment. The reported biodegradation half-lives for acenaphthylene in anaerobic soil range from 12 to 121 days. Acenaphthylene is not expected to hydrolyze or bioconcentrate in the environment but it will undergo direct photolysis in sunlit environmental media. A calculated Koc range of 950 to 3315 indicates acenaphthylene will have a low to slight mobility in soil. In aquatic systems, acenaphthylene

may partition from the water column to organic sediments and suspended solids. A Henry's Law constant of 1.13X10-5 atm-cu m/mole at 25° C suggests volatilization of acenaphthylene from surface waters may be significant fate process. The volatilization half-lives from a model river and a model pond (the latter considers the effect of adsorption) have been estimated to be 4 and 184 days, respectively. Acenaphthylene is expected to exist entirely in the vapor-phase in ambient air. In the atmosphere, reactions with photochemically produced hydroxyl radicals and ozone (respective estimated half-lives of 5 and 1 hours) are likely to be important fate processes (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. No bioconcentration/bioaccumulation data was available for this compound.

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

No compound-specific information could be found on the toxic effects of acenapthylene on mammals or aquatic organisms, or birds.

E.2.3.4   Anthracene

Anthracene is a ubiquitous product of incomplete combustion; sources include exhaust from gasoline and diesel engines, cigarette smoke, emissions from coal, oil, and wood burning stoves, furnaces and power plants. In the environment anthracene tends to biodegrade rapidly but does not hydrolyze. The reported half-lives for soils ranged from 3.3 to 139 days. Anthracene will adsorb very strongly to soil and thus will not migrate significantly into the groundwater. When anthracene is present near the soil surface it may also evaporate. Adsorption to soil will be expected to retard both evaporation and biodegradation processes. In aquatic systems anthracene tends to bind to sediments and particulate matter. As in the soils, in aquatic environments anthracene will biodegrade but will not hydrolyze. It will also be subject to direct photolysis near the water surface. In moving water the half-live of anthracene was predicted to range between 4.3 to 5.9 days. Adsorption of anthracene to sediments, and particulates may retard the evaporation, biodegradation, bioconcentration, and photolysis processes. In the atmosphere, anthracene will be subject to direct photolysis and the estimated vapor phase half-life in the atmosphere is 1.67 days as a result of reaction with photochemically produced hydroxyl radicals (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify this compound in the food chain due to their ability to rapidly metabolize PAHs. In addition, bioconcentration factors for anthracene tended to decrease with Aldrich humic acids (HSDB 1997). However, certain organisms which lack the PAH-metabolizing enzymes may bioaccumulate this compound to some extenet. Below is a list of bioaccumulation/bioconcentration rates reported by Eisler (1987b) and HSDB (1997) for this compound:

Taxonomic Group	BCF/BAF	Exposure Period
Cladoceran (*D. magna*)	200	60 months
Fathead Minnow (*P. promelas*)	485	2 to 3 days
Cladoceran (*D. magna*	760 to 1,200	1 day
Mayfly (*Hexagenia* sp.)	3,500	28 hours
Rainbow trout (*Salmo Gairdneri*)	4,400 to 9,200	72 hours
Benthic invertebrate (*P. hoyi*)	16,800	48 hours
Bluegills (*L. macrochirus*)	900	NA

No toxic mechanisms other than those described above for PAHs were located in the literature for this compound.

Very limited data was available evaluating the toxic effects of anthracene to mammals. A chronic oral dose of 3,300 mg/kg BW was carcinogenic to rodents (Eisler 1987b). A lethal dose of anthracene to mice was 17,000 mg/kg. Mice exposed to anthracene in this study also showed fatty liver degeneration (RTECS 1997). Anthracene was administered to groups of 20 male and female mice by oral gavage at doses of 0, 250, 500, and 1000 mg/kg/day for at least 90 days. Mortality, clinical signs, body weights, food consumption, opthalmology findings, hematology and clinical chemistry results, organ weights, organ-to-body weight ratios, gross pathology, and histopathology findings were evaluated. No treatment-related effects were noted at any concentration evaluated. (IRIS 1997).

Several studies evaluating the toxic effect to aquatic organisms were obtained for anthracene. Most studies were focused on photo toxic effects of anthracene. Acute mortality of bluegill sunfish (*Lepomis macrochirus*) dosed with anthracene at 12.7 micrograms (μg/L) and exposed to natural sunlight conditions was observed during a study of anthracene fate in outdoor channel microcosms. No mortality was observed under control conditions (natural sunlight and no anthracene). Fish survived when held in the shade downstream of sunlit contaminated water, arguing against mortality due to toxic anthracene photo-products in the water. Fish held 48 hours in anthracene contaminated water (12 μg/L) in a shaded channel died when placed in clean water and exposed to sunlight. After a similar exposure period, followed directly by 144 hours depuration in darkness, fish anthracene concentrations had decreased to preexposure concentrations, and no mortality was observed when fish were subsequently exposed to sunlight (HSDB 1997).

Protozoans (*Paramecium caudatum*) exposed to 0.1 μg/L of anthracene for 60 minutes, exhibited a 90% lethal photodynamic response. Photoinduced anthracene toxicity to Daphnia (*Daphnia pulex*) was investigated using organisms that were exposed to 3 concentrations of anthracene (3.0, 9.6, and 30 μg/L) in static bioassays on clear, partly cloudy, and cloudy days. Photoinduced anthracene toxicity was not observed under laboratory lighting conditions; it occurred only in the presence of solar radiation. A dose response relation was observed for both anthracene concentration and solar radiation intensity (HSDB 1997).

Acute toxicity concentrations from phototoxicity studies were also available in the literature for several additional species. The 24-hour LC50 Culicid mosquito larvae was 26.8 μg/L, the 96-hour LC50 juvenile bluegills (*Lepomis macrochirus*) was 11.9 μg/L, and the 5-hour LC50 for leopard frogs (*Rana pipiens*) was 0.065 ppm (HSDB 1997).

No compound-specific information could be found on the toxic effects of anthracene on birds.

E.2.3.5   Fluorene

Fluorene occurs in fossil fuels. Its release to the environment is wide spread since it is a common product of incomplete combustion. It is released to the atmosphere in emissions from the combustion of oil, gasoline, coal, wood, and refuse. If released to the atmosphere, fluorene will exist primarily in the vapor phase where it will degrade readily by photochemically produced hydroxyl radicals (estimated half-life of 29 hours). In soil or water, fluorene will biodegrade readily (aerobically) in the presence of acclimated microbes; microbial adaptation is an important fate process. Biodegradation can be slow in pristine soils or waters or under conditions of limited oxygen. Strong adsorption to soil and water

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sediment is an important transport process. The half-life of fluorene in soil has been reported to range from 2 to 64 days (HSDB 1997). Volatilization of non-adsorbed fluorene from water may be important process with estimated half-lives of 15 and 167 hours from a model river and model pond respectively (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. Below is a list of bioaccumulation/bioconcentration rates reported by Eisler (1987b) and HSDB (1997) for this compound:

Taxonomic Group	BCF/BAF	Exposure Period
Fathead minnow (P. promelas)	1,480	28 days
Guppy Fish (Poecilia reticulata)	1,050 to 2,240	2 to 4 days
Mosquitofish (Gambusia affinis)	1,410	33 days

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

Mice (25/sex/group) were exposed by gavage to 0, 125, 250, or 500 mg/kg/day fluorene suspended in corn oil for 13 weeks. Parameters used to assess toxicity included food intake, body weight, clinical observations, hematology and serum chemistry and gross and histopathological examinations. Increased salivation, hypoactivity, and urine-wet abdomens in males were observed in all treated animals. The percentage of mice exhibiting hypoactivity was dose-related. In mice exposed at 500 mg/kg/day, labored respiration, ptosis (drooping eyelids), and unkempt appearance were also observed. A significant decrease in red blood cell count and packed cell volume were observed in females treated with 250 mg/kg/day fluorene and in males and females treated with 500 mg/kg/day. Decreased hemoglobin concentration and increased total serum bilirubin levels were also observed in the 500 mg/kg/day group. Decreases in erythrocyte count, packed cell volume, and hemoglobin concentration were all observed at 125 mg/kg; however, these effects, although apparently dose-dependent, were not statistically significant. A significant decreasing trend in BUN and a significant increasing trend in total serum bilirubin were observed in both high-dose males and females. A dose-related increase in relative liver weight was observed in treated mice; a significant increase in absolute liver weight was also observed in the mice treated with 250 and 500 mg/kg/day fluorene. A significant increase in absolute and relative spleen and kidney weight was observed in males and females exposed to 500 mg/kg/day and males at 250 mg/kg/day. Increases in the absolute and relative liver and spleen weights in the high-dose males and females were accompanied by histopathological increases in the amounts of hemosiderin in the spleen and in the Kupffer cells of the liver. No other histopathological lesions were observed (IRIS 1997).

Static aquatic toxicity tests were conducted on daphnids (Daphnia magna), larval midges (Chironomus riparius), amphipods (Gammarus pseudolimnaeus), snails (Mudalia potosensis), mayflies (Hexagenia bilineata), bluegill (Lepomis macrochirus), rainbow trout (Salmo gairdneri), fathead minnows (Pimephales promelas), aquatic macrophytes (Chara sp), and green algae (Selanastrum capricornutum). Daphnia magna was the most sensitive organism tested with a 48 hours median effective concentration of 0.43 mg/l. Fathead minnows were the least sensitive species, with no mortality at fluorene concentrations as high as 100 mg/l. In a 14 day test, fluorene exposure inhibited algal production at a threshold level of approximately 3.0 mg/l. Complete life cycle chronic toxicity tests were conducted with fluorene on daphnids and larval midges. Daphnid reproduction was significantly reduced at fluorene levels of 0.125 mg/l after 14 days. Emergence of larval midges was delayed at a concentration of 0.6 mg/l. In a 30 day partial life cycle study conducted to

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determine the impact of fluorene on growth, survival, and behavior of fingerling bluegill (*Lepomis macrochirus*), survival was reduced at exposures of 0.5 and 1.0 mg/l, and growth was inhibited at exposures of 0.25, 0.5, and 1.0 mg/l. Measurements of several behavioral characteristics indicated impairment of swimming and feeding activities at fluorene concentrations as low as 0.12 mg/l (HSDB 1997).

No compound-specific information could be found on the effects of fluorene on birds.

E.2.3.6   Naphthalene

Naphthalene enters the atmosphere primarily from fugitive emissions and exhaust connected with its presence in fuel oil and gasoline. In addition, there are discharges on land and into water from spills during the storage, transport and disposal of fuel oil, coal tar, and other petroleum products. Naphthalene is the most abundant single constituent of coal tar (Budavari et al. 1989). Once in the atmosphere, naphthalene rapidly photodegrades (half-life 3-8 hours). Naphthalene released into the water column is lost due to volatilization, photolysis, adsorption to sediment and particulate matter, and biodegradation. The principal loss processes will depend on local conditions, but half-lives can be expected to range from a couple of days to a few months. When adsorbed to sediment, biodegradation occurs much more rapidly than in the overlying water column. Naphthalene is adsorbed moderately to soil and undergoes biodegradation. Evaporation of naphthalene from the top soil layer will be important but the importance of the process will gradually decrease as the soil depth increases. However, in some cases it will appear in the groundwater where biodegradation still may occur if conditions are aerobic (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. Below is a list of bioaccumulation/bioconcentration rates reported by Eisler (1987b) and HSDB (1997) for this compound:

Taxonomic Group	BCF/BAF	Exposure Period
Sandworm (*Neanthes arenaceodenta*)	40	1 day
Sandworm (*Neanthes arenaceodenta*)	Not detectable	1 day exposure and 300 days post-exposure
Clam (*Rangia cuneata*)	6	1 day
Cladoceran (*Daphnia magna*)	131	1 day
Crustaceans (Average of 3 species)	195 to 404	72 hour
Bluegill (*Lepomis macrochirus*)	310	1 day

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

Several studies were available in the literature documenting the toxic effects of naphthalene to mammalian species. Pregnant rabbits were gavaged with 16 mg/kg of metabolite of naphthalene on days 20, 22, and 24 of gestation. Cataracts and retinal damage were found in the offspring. Dogs administered 420 and 1,530 mg/kg naphthalene (in a solid form) in a single oral dose showed decreases of 29 and 33%, respectively, in blood hemoglobin concentrations. Male and female mice were dosed with 267 mg/kg/day of naphthalene for 14 days. Effects observed included increased mortality, decreased terminal body weights, a decrease in absolute thymus weight (30 percent) in males, and an increase bilirubin levels and a decrease in absolute and relative spleen and lung weights compared to controls in females. No carcinogenic response was observed in rats given oral doses of 10 to 20 mg/day naphthalene, 6 days/week from day 100 to day 800 of age. The median lethal dose (LD50s)

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were available for several species of rats and mice. The LD50s ranged from 533 mg/kg in male CD-1 mice (gavage) to 2,600 mg/kg in Sprague Dawley rats (oral) (HSDB 1997).

For aquatic species, LC50s ranged from 2,000 mg/L in dungeness crab (*Cancer magister*) to 150,000 mg/L in mosquitofish (*Gambusia affinis*). Other species evaluated [including grass shrimp, amphipods (*Elasmopus pectenicrus*), coho salmon (*Oncorhyncus kisutch*), and sandworms] all had LC50s between 2,400 and 3,800 mg/L (Eisler 1987b). The 96-hour Median Threshold Limits (TLms) for pink salmon (*Onchorynchus gorbuscha*) and shrimp (*Pandalus goniurus*) were 1.2 mg/L and 0.97 mg/L at 12° C, respectively (HSDB 1997).

Larval mud crabs were exposed continuously from hatching through 1st stage to sublethal concentrations of naphthalene (0, 75, 150 or 300 mg/L). During the study, salinity and temperature levels varied but at optimal salinity levels no consistent effect of naphthalene on growth was observed (HSDB 1997).

No compound-specific information could be found on the toxic effects of naphthalene on birds.

E.2.3.7 Phenanthrene

Phenanthrene is a component of the incomplete combustion of a variety of organic compounds including wood and fossil fuels. Phenanthrene was detected in used motor oil (158 mg/kg), crude oils (129 mg/kg), and lubricating fuels (7.1 mg/kg). In soils phenanthrene will likely biodegrade, and volatilization is not expected to be significant. Phenanthrene strongly binds to soil and should not leach extensively to groundwater. When released to water, adsorption of phenanthrene to suspended sediments is expected to remove most of the compound from solution. Photolysis is expected to occur near the water surface, and biodegradation of phenanthrene in the water column is expected. Oxidation and volatilization are not expected to be significant. Phenanthrene released to the atmosphere is expected to rapidly adsorb to particulate matter (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. Below is a list of bioaccumulation/bioconcentration rates reported by Eisler (1987b) and HSDB (1997) for this compound:

Taxonomic Group	BCF/BAF	Exposure Period
Clam (*Rangia cuneata*)	32	1 day
Cladoceran (*Daphnia pulex*)	325	1 day

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

Limited data on the effects of phenanthrene on mammals was available in the literature. Phenanthrene rarely caused tumors or papillomas when ingested or applied directly to the skin of rats and mice. The oral LD50 in mice was calculated to be 700 mg/kg (Eisler 1987b). Rats injected with a single intraperitoneal injection of phenanthrene at a concentration of 150 mg/kg BW had altered blood serum chemistry and nephrotoxicity (Eisler 1987b).

No information on chronic toxicity was obtained from the literature for any aquatic organisms. The marine snail, (*Littorina littorea*), was exposed to phenanthrene. Phenanthrene was added each day for 3 days at a concentration of 40 mg/L. Digestive cell

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lysosomal labilization as measured by beta-glucuronidase and arylsulfatase was used as a measure of toxicity. Snails exposed to the phenanthrene exhibited a pronounced lysosome labilization compared to the control group (HSDB 1997). A 24-hour LC50 of 370 µg/L was reported for grass shrimp and a 96-hour LC50 of 600 µg/kg was reported for sandworms (Eisler 1987b).

No compound-specific information could be found on the toxic effects of phenanthrene on birds.

E.2.3.8 Benzo(a)anthracene

Benzo(a)anthracene is a universal product of combustion of organic matter. Typical sources of benzo(a)anthracene are engine exhaust, cigarette smoke, coal-tar pitch, coke oven emissions, and soot and smoke of industrial and domestic origin. Benzo(a)anthracene is a produced by the combustion of organic matter. Its production is favored by oxygen-deficient flames with temperatures in the range of 650-900° C and by fuels which are not highly oxidized. In both air and water benzo(a)anthracene is typically largely associated with particulate matter. In the water column benzo(a)anthracene will rapidly become adsorbed to sediment or particulate matter in the water column. In the unadsorbed state it will degrade by photolysis in a matter of hours to days, while adsorbed there is little evidence of photodegradation. Benzo(a)anthracene typically will not leach into the groundwater and will remain in the upper few centimeters of soil since it is strongly adsorbs to soil particles. Benzo(a)anthracene will very slowly biodegrade when colonies of microorganisms are acclimated but this is a slow process (half-life about 1-year). Benzo(a)anthracene in the atmosphere will be transported long distances and will probably be subject to photolysis and photooxidation although there is little documentation about the rate of these processes in the literature (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. Below is a list of bioaccumulation/bioconcentration rates reported by Eisler (1987b) and HSDB (1997) for this compound:

Taxonomic Group	BCF/BAF	Exposure Period
Cladoceran (*Daphnia pulex*)	10,109	1 day

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

Limited data on the effects of benzo(a)anthracene on mammals was available in the literature. A chronic oral dose of 2.0 mg/kg BW per day was carcinogenic to rodents (Eisler 1987b).

A 6-month LC87 value for bluegill (*Lepomis macrochirus*) is 1,000 µg/L (Eisler 1987b).

No compound-specific information could be found on the toxic effects of benzo(a)anthracene on birds.

E.2.3.9 Benzo(a)pyrene

The release of Benzo(a)pyrene to the environment is widespread since it is an ubiquitous product of incomplete combustion. It is largely associated with particulate matter, soils, and sediments. Although environmental concentrations are highest near sources, its presence in

places distant from primary sources indicates that it is reasonably stable in the atmosphere and capable of long distance transport. When released to air it may be subject to direct photolysis, although adsorption to particulates apparently can retard this process. In the water, benzo(a)pyrene will adsorb very strongly to sediments and particulate matter, bioconcentrate in aquatic organisms which can not metabolize it, but will not hydrolyze. It may be subject to significant biodegradation, and direct photolysis may be important near the surface of waters; adsorption, however, may significantly retard these two processes. Evaporation may be important but again adsorption to sediments and particulates will limit evaporative rates. If released to soil it will be expected to adsorb very strongly to the soil; it will not be expected to appreciably leach to groundwater, hydrolyze, or evaporate. It may be subject to appreciable biodegradation in soils, especially in preexposed soils.

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. Below is a list of bioaccumulation/bioconcentration rates reported by Eisler (1987b) and HSDB (1997) for this compound:

Taxonomic Group	BCF/BAF	Exposure Period
Algae (*Oedogonium cardiacum*)	5,258	3 day
Clam (*Rangia cuneata*)	9 to 236	1 day
Oyster (*Crassostrea virginica*)	242	14 day
Snail (*Physa* sp.)	82,231	3 day
Cladoceran (*Daphnia magna*)	2,837	6 hour
Cladoceran (*Daphnia magna*)	134,248	3 day
Midge (*Chironomus riparius*), larvae	166	8 hour
Bluegill (*Lepomis macrochirus*)	12	4 hour
Mosquitofish (*Gambusia affinis*)	930	3 day

In certain species dietary ingestion of PAHs has been shown to be an important route of exposure. In a laboratory aquatic ecosystem study, Lu et al. (1977) determined that almost all of the benzo(a)pyrene accumulated by mosquitofish (*Gambusia affinis*) was through dietary ingestion with negligible accumulations from the medium (i.e., water).

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

Benzo(a)pyrene has been shown to cause a variety of adverse reproductive and carcinogenic effects in mammals. Oral administration of 10 mg/kg BW/day of benzo(a)pyrene to CD-1 mice during pregnancy resulted in a reduction of gonadal weight in the offspring but had no significant effect on body weight of offspring. These exposure levels also resulted in a reduction of fertility and reproductive capacity. At a dose of 40 mg/kg BW/day during pregnancy almost complete sterility was observed in offspring of both sexes. In another study, rats fed 1 mg of benzo(a)pyrene per gram of diet during pregnancy resulted in many resorptions and dead fetuses in the offspring, although only one malformed fetus was observed in 7 litters (HSDB 1997). Sims and Overcash (1978) evaluated acute toxicity of benzo(a)pyrene in mice and rats. Of the four PAH compounds evaluated benzo(a)pyrene had the lowest reported LD50, 50 mg/kg BW/day. The LD50s for the other PAH compounds (phenanthrene, naphthalene, and fluoranthene) were between 700 and 2,000 mg/kg BW/day. A chronic oral dose of 0.002 mg/kg BW/day was determined to be carcinogenic to rodents (Eisler 1987b).

Rainbow trout alevins were reared in a range of aqueous benzo(a)pyrene concentrations (0.00, 0.08, 0.21, 0.39, 1.48, 2.40, or 2.99 ng/mL). At the conclusion of the study

histological and skeletal examinations were performed. Nuclear pycnosis and karyorrhexis were most common in neuroectodermal and mesodermal derivatives and in liver of benzo(a)pyrene-treated alevins. Microphthalmia was noted in 17% of the test fish and was frequently associated with a patent optic fissure. Depressed mitotic rates in the retina and brain, but not liver, were seen in alevins reared in 0.21 to 1.48 ng/ml aqueous benzo(a)pyrene. Test alevins had a significantly higher incidence of skeletal malformations in the skull and vertebral column and abnormalities of vertebral arcualia often corresponded to areas of kyphoscoliotic flexures. The ecological significance of such morphological abnormalities would be decreased feeding and growth and inability to escape predation, leading to reduced survival. A 96-hour LC50 value for sandworms (*Neanthes arenceodentata*) exceed the highest test dose of 1,000 µg/L (Eisler 1987b).

No compound-specific information could be found on the toxic effects of benzo(a)pyrene on birds.

E.2.3.10   Benzo(b)fluoranthrene

Benzo(b)fluoranthene is typically formed as a result of the incomplete combustion of a variety of fuels including wood and fossil fuels. When released to water, adsorption to suspended particles and sediments is expected to remove most of the benzo(b)fluoranthene from solution. Photolysis and photooxidation of the benzo(b)fluoranthene which remains in solution is expected to occur, but adsorbed benzo(b)fluoranthene is expected to resist these processes. Volatilization and biodegradation of dissolved benzo(b)fluoranthene may also occur in aquatic systems. When released to the soil some biodegradation and volatilization may also occur. Due to the anticipated strong adsorption to the soil, these processes occur at very slow rates and are considered to be relatively insignificant processes. Benzo(b)fluoranthene in the atmosphere is likely to be adsorbed to particulate matter, and will be subject to wet and dry deposition. Benzo(b)fluoranthene in the vapor phase will react with photochemically generated, atmospheric hydroxyl radicals with an estimated half-life of 1 day. Photolysis of vapor phase benzo(b)fluoranthene will be rapid, but the adsorbed compound may not photolyze significantly.

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. No bioconcentration/bioaccumulation data was available for this compound.

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

No compound-specific information could be found on the toxic effects of benzo(a)fluoranthrene on mammals, aquatic organisms, or birds.

E.2.3.11   Benzo(g,h,i)perylene

Benzo(g,h,i)perylene is a component of crude oil and a product of the incomplete combustion of a variety of organic compounds including wood and fossil fuels. Emissions from petroleum refining, coal tar distillation, and the combustion of wood, coal, oil, propane, gasoline and diesel fuels are major contributors of benzo(g,h,i)perylene to the environment. Benzo(g,h,i)perylene is not commercially produced in the USA; however, benzo(g,h,i)perylene may be released to the environment through industrial effluents, municipal waste water treatment facilities and waste incinerators. Benzo(g,h,i)perylene biodegrades slowly in the environment. The reported biodegradation half-lives for benzo(g,h,i)perylene in aerobic soil range from 600 to 650 days. Benzo(g,h,i)perylene is not

expected to hydrolyze in the environment. A calculated Koc range of 90,000 to 400,000 indicates benzo(g,h,i)perylene will be highly immobile in soil, limiting its leaching into groundwater. In aquatic systems, benzo(g,h,i)perylene partitions from the water column to organic matter contained in sediments and suspended solids. The volatilization half-lives from a model river and a model pond (the latter considers the effect of adsorption) have been estimated to be 38 days and over 1,500 years, respectively. In the atmosphere, the vapor phase reaction with photochemically produced hydroxyl radicals (half-life of 2 hours) may be an important fate process. Benzo(g,h,i)perylene may also undergo direct photolysis in the atmosphere. However, benzo(g,h,i)perylene is expected to exist almost entirely in the particulate phase in ambient air. In the atmosphere, adsorption to stabilizing substrates will allow benzo(g,h,i)perylene to be transported over long distances in the atmospheric aerosol. Its presence in lake sediments in the Adirondack Forest, NY, has been attributed to physical deposition. Removal of adsorbed benzo(g,h,i)perylene from the atmosphere may occur by wet and dry deposition (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. No bioconcentration/bioaccumulation data was available for this compound.

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

The physiological effects of a single ingested dose of Prudhoe bay crude oil, its aromatic fractions, and Prudhoe bay crude corexit 9527 emulsion were studied in nestling herring gulls (*Larus argentatus*). The high molecular weight aromatic compounds, including benzo(g,h,i)perylene, were responsible for retardation of growth and increases in adrenal and nasal gland weight (HSDB 1997).

No compound-specific information could be found on the toxic effects of benzo(g,h,i)perylene on mammals or aquatic organisms, or birds.

E.2.3.12   Benzo(k)fluoranthene

Benzo(k)fluoranthene is a component of crude oil and a product of the incomplete combustion of a variety of organic compounds including wood and fossil fuels. Emissions from petroleum refining, coal tar distillation, and the combustion of wood, coal, oil, propane, gasoline and diesel fuels are major contributors of benzo(k)fluoranthene to the environment. Benzo(k)fluoranthene is also released to the environment through industrial effluents, municipal waste water treatment facilities and waste incinerators. Benzo(k)fluoranthene biodegrades slowly in the environment. In soils benzo(k)fluoranthene adsorbs strongly to the soil particles and remains in the upper layers and with little to no leaching into groundwater. Biodegradation may also occur but will be very slow (half-life about 2 years in soils with acclimated microorganisms). Benzo(k)fluoranthene is not expected to hydrolyze in the environment. In aquatic systems, benzo(k)fluoranthene partitions from the water column to organic matter contained in sediments and suspended solids. In the atmosphere, the vapor phase reaction with photochemically produced hydroxyl radicals (half-life of 2 hours) may be an important fate process. Benzo(k)fluoranthene may also undergo direct photolysis in the atmosphere. However, benzo(k)fluoranthene is expected to exist primarily in the particulate phase in ambient air. Removal of adsorbed benzo(k)fluoranthene from the atmosphere may occur by wet and dry deposition.

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. No

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bioconcentration/bioaccumulation data was available for this compound.

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

No compound-specific information could be found on the toxic effects of benzo(k)fluoranthrene on mammals or aquatic organisms, or birds.

E.2.3.13   Chrysene

Chrysene is a component of crude oil and a product of the incomplete combustion of a variety of organic compounds including wood and fossil fuels.   In terrestrial and aquatic systems, chrysene is largely associated with particulate matter, soils, and sediments. If released to soil it will be expected to adsorb very strongly to the soil and will not be expected to leach appreciably to groundwater. It will not hydrolyze or appreciably evaporate from soils or surfaces, and it may be subject to biodegradation in soils. If released to water, it will adsorb very strongly to sediments and particulate matter, but will not hydrolyze or appreciably evaporate.   Based on limited available data, chrysene may be subject to biodegradation in aquatic systems. Adsorption to various materials may decrease the rate of these processes. If released to air, chrysene will be subject to direct photolysis, although adsorption to particulates may limit the rate of this process. The estimated half-life of any gas phase chrysene in the atmosphere is 1.25 hrs as a result of reaction with photochemically produced hydroxyl radicals (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. Below is a list of bioaccumulation/bioconcentration rates reported by Eisler (1987b) and HSDB (1997) for this compound:

Taxonomic Group	BCF/BAF	Exposure Period
Clam (*Rangia cuneata*)	8	1 day
Pink Shrimp (*P. duorarum*), Cephalothorax	248 to 361	28 day
Pink Shrimp (*P. duorarum*), Cephalothorax	21 to 48	28 days post-exposure and 28 days exposure

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

The only toxicity data available for the effects of chrysene on aquatic systems was a sandworm acute toxicity test. The ninety-six hour LD50 exceeded the highest test concentration, 1,000 µg/L (Eisler 1987b).

No compound-specific information could be found on the toxic effects of chrysene on mammals or birds.

E.2.3.14   Dibenz(a,h)anthracene

Dibenz(a,h)anthracene is a component of crude oil and a product of the incomplete combustion of a variety of organic compounds including wood and fossil fuels. In the environment dibenz(a,h)anthracene is associated with particulate matter, including soils, sediments and, suspended solids. Its presence in places distant from primary sources indicates that it is reasonably stable in the atmosphere and capable of long distance transport. In soils dibenz(a,h)anthracene is expected to adsorb very strongly to the soils and

will not typically leach to the groundwater, hydrolyze or evaporate. In soils it will be subject to biodegradation with reported half-lives ranging between 18 and 21 days. In aquatic systems, dibenz(a,h)anthracene will adsorb very strongly to sediments and particulate matter. Based on limited data from laboratory screening tests using settled domestic wastewater and activated sludge, dibenz(a,h)anthracene may be subject to biodegradation in natural waters. Since dibenz(a,h)anthracene adsorbs solar radiation strongly, it may also be subject to direct photolysis in natural waters. Adsorption to sediments may significantly retard these degradation processes. Hydrolyzation and evaporation are not considered to be significant fate processes. In the atmosphere it will likely be associated with particulate matter and may be subject to moderately long range transport, depending mainly on the particle size distribution and climatic conditions which will determine the rates of wet and dry deposition. Dibenz(a,h)anthracene may be subject to direct photolysis in the atmosphere; however, adsorption may significantly retard the rate of photolysis. The estimated vapor phase half-life in the atmosphere is 1 day as a result of reaction with photochemically produced hydroxyl radicals (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. Below is a list of bioaccumulation/bioconcentration rates reported by Eisler (1987b) and HSDB (1997) for this compound:

Taxonomic Group	BCF/BAF	Exposure Period (Exposure Conc.)
Cladoceran (*Daphnia magna*)	652	Unknown (0.2 mg/kg Aldrich humic acids added)
Cladoceran (*Daphnia magna*)	773	Unknown (2.0 mg/kg Aldrich humic acids added)

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

Forty-two mice receiving 0.2 mg/mL of olive oil emulsion in place of drinking water at a average dose of 0.76 to 0.85 mg/day showed a high incidence of pulmonary adenomatosis, alveologenic carcinoma of the lung, hemangio-endothliomas, and mammary carcinomas compared to the controls (HSDB 1997). No additional chronic or acute toxicity data was available in the literature for mammals.

A median threshold limit (TLm) of 1 mg/L was calculated for *Neanthes arenceodentata* in a 96-hour static bioassay (HSDB 1997). The LD50 for sandworms exceeded the highest test concentration, 1,000 µg/L, in a 96-hour bioassay (Eisler 1987b).

No compound-specific information could be found on the toxic effects of dibenz(a,h)anthracene on birds.

E.2.3.15 Fluoranthene

Fluoranthene is a component of crude oil and a product of the incomplete combustion of a variety of organic compounds including wood and fossil fuels. Its release is greatest in areas of high anthropogenic activity. In the environment fluoranthene is associated with particulate matter, including soils, sediments and, suspended solids. In aquatic system, fluoranthene will rapidly become adsorbed to sediment and particulate matter in the water column. In the unadsorbed state it will degrade by photolysis (half-life days to week). It appears to be stable in sediment for decades or more. In terrestrial systems, it will strongly adsorb to soils and remain in the upper few centimeters of the soil limiting its leaching to

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groundwater. Fluoranthene will typically biodegrade in a soils, with the most rapid rates occurring when in the soil contains acclimated microorganisms. The fluoranthene released in the atmosphere will photodegrade in the free state (half-life 4-5 days). Aerosols and particulate matter containing sorbed fluoranthene is sufficiently stable to be transported long distances while being subject to wet and dry deposition (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. Below is a list of bioaccumulation/bioconcentration rates reported by Eisler (1987b) and HSDB (1997) for this compound:

Taxonomic Group	BCF/BAF	Exposure Period
Fathead minnow (Pi*mephales promelas*)	3,980	7 days
Rainbow trout (*Salmo Gairdneri*)	380	21 days
Oysters	12,300	2 days

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

Male and female mice (20/sex/group) were exposed by gavage for 13 weeks with 0, 125, 250, or 500 mg/kg/day fluoranthene. A fifth group of mice (30/sex) was established in the study for baseline blood evaluations. Body weight, food consumption, and hematological and serum parameter values were recorded at regular intervals during the experiment. At the end of 13 weeks, the animals were sacrificed, organ weights were measured, and a histopathological evaluation was performed. All treated mice exhibited nephropathy, increased salivation, and increased liver enzyme levels in a dose-dependent manner. However, these effects were either not significant, not dose-related, or not considered adverse at 125 mg/kg/day. Mice exposed to 500 mg/kg/day had increased food consumption and increased body weight. Mice exposed to 250 and 500 mg/kg/day had statistically increased serum glucose phosphate transferase (SGPT) values and increased absolute and relative liver weights. Compound-related microscopic liver lesions (indicated by pigmentation) were observed in 65 and 87.5% of the mid- and high-dose mice, respectively (IRIS 1997). The oral LD50 in rats was calculated to be 2,000 mg/kg (RTECS 1997).

In aquatic systems, LC50 concentrations for 96-hour toxicity tests were available for mysid shrimp (*Mysidopsis bahia*), bluegills (*Lepomis macrochirus*), polycheates, and sheepshead minnows (*Cyprinodon variegatus*). The sensitivities of three organisms to fluoranthene varied considerably. Mysid shrimp had the lowest LC50 (0.04 mg/L) followed by bluegills (4.0 mg/L), polycheates (500 mg/L), and sheepshead minnows (560 mg/L). The Median Ecological Effects (EC50s) for alga (*Selenastrum capricornutum*) for 96-hour bioassays were 55 and 46 mg/L for effects on chlorophyll and effects on cell numbers, respectively.

No compound-specific information could be found on the toxic effects of fluoranthene on birds.

E.2.3.16 Indeno(1,2,3-cd)pyrene

Indeno(1,2,3-cd)pyrene is formed during most combustion or elevated temperature processes that involve compounds containing carbon and hydrogen (e.g., coal, wood, and gasoline combustion, municipal waste incineration, coke ovens, and cigarette smoke). Indeno(1,2,3-cd)pyrene has also been found in gasoline, fresh and used motor oil, and road runoff. Indeno(1,2,3-cd)pyrene released to soil will sorb strongly (estimated Koc = 20,146) and hence is not expected to leach into the groundwater. No information was located

regarding volatilization, hydrolysis, or biodegradation of indeno(1,2,3-cd)pyrene terrestrial or aquatic systems. In aquatic systems, indeno(1,2,3-cd)pyrene will sorb strongly to suspended particulate matter and sediments. Almost all indeno(1,2,3-cd)pyrene released to the atmosphere will be sorbed to particulate matter; thus its atmospheric fate will primarily depend on physical processes such as dry and wet deposition (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. No bioconcentration/bioaccumulation data was available for this compound.

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

No compound-specific information could be found on the toxic effects of indeno(1,2,3-cd)pyrene on mammals, aquatic species, or birds.

E.2.3.17   Pyrene

Pyrene is a common product of incomplete combustion, largely associated with particulate matter, soil, and sediment. It is reasonably stable when adsorbed by particulate matter in the atmosphere and capable of long distance transport. In water, it adsorbs readily to sediment and particulate matter, but does not hydrolyze. It may be subject to significant biodegradation; direct photolysis may be important near the water surface. It is expected to adsorb very strongly to soil and not appreciably leach to the ground water. It is not expected to significantly evaporate from or hydrolyze in soil (HSDB 1997).

As stated previously, most mammals, fish and crustaceans will not tend to biomagnify PAHs in the food chain due to their ability to rapidly metabolize PAHs. Below is a list of bioaccumulation/bioconcentration rates reported by Eisler (1987b) and HSDB (1997) for this compound:

Taxonomic Group	BCF/BAF	Exposure Period
Cladoceran (*Daphnia pulex*)	2702	1 day
Rainbow trout (*Salmo Gairdneri*)	72	Not known
Fathead minnow (*P. promelas*)	600 to 970	Not known

No toxic mechanisms other than those described above for PAHs were available in the literature for this compound.

Male and female mice (20/sex/group) were exposed by gavage to 0, 75, 125, or 250 mg/kg/day pyrene in corn oil for 13 weeks. The toxicological parameters examined in this study included body weight changes, food consumption, mortality, clinical pathological evaluations of major organs and tissues, and hematology and serum chemistry. Nephropathy, characterized by the presence of multiple foci of renal tubular regeneration, often accompanied by interstitial lymphocytic infiltrates and/or foci of interstitial fibrosis, was present in 4, 1, 1, and 9 male mice in the control, low-, medium-, and high-dose groups, respectively. Similar lesions were seen in 2, 3, 7, and 10 female mice in the 0, 75, 125, and 250 mg/kg treatment groups. The kidney lesions were described as minimal or mild in all dose groups (IRIS 1997). The oral LD50 in rats and mice were calculated to be 2,700 and 800 mg/kg, respectively. Toxic effects observed in both mice and rats include conjunctive irritation, excitement, and muscle contraction or spasticity (RTECS 1997).

A median threshold limit (TLm) of 0.0026 mg/L/96 hours at 24 to 27° C in a static bioassay was reported for the mosquito fish (HSDB 1997).

No compound-specific information could be found on the toxic effects of pyrene on birds.

E.3   Pesticides

E.3.1   DDT

DDT and its metabolites (referred to collectively in this section as DDTr) are hydrophobic, and thus would not be expected to be present in surface waters at high concentrations. The majority of DDTr entering aquatic systems is expected to accumulate in sediments and biological tissues. DDT and its metabolites are known to accumulate in biological tissues, particularly lipids, where they may be stored for extended periods of time and be passed on to higher trophic level organisms. Several studies have indicated that DDTr biomagnifies, or is found in biological tissues at increasing concentrations at higher trophic levels. Biologically accumulated DDT (or its metabolites) may be metabolized to another form (i.e., DDT may be transformed to DDE). When fat reserves are metabolized, the DDT or transformed metabolite is released into the system, where it may result in a toxic response. DDTr may act as a direct toxin to some receptors; however, because of its tendency to concentrate in biological tissues, higher trophic level receptors may be at increased risk through ingestion of contaminated food sources.

DDT and its metabolites appear to affect the reproductive success of many receptors. One well documented response is eggshell thinning in birds exposed to p,p'-DDE, which affects the activity of Ca^{2+} ATP-ase systems in the shell gland, thereby interfering with the deposition of calcium in the shell (Lundholm 1987; Lundholm 1988; Miller $et\ al$. 1976). Eggshell thinning of greater than 20 percent has been associated with decreased nesting success due to eggshell breakage (Anderson and Hickey 1972; Dilworth $et\ al$. 1972). Because of tendency of DDTr to magnify in food chains, higher trophic level birds (i.e., piscivorous, raptors) appear to be at greater risk for egg loss due to shell thinning.

Another well defined effect of DDTr exposure is inhibition of acetylcholinesterase (AChE) activity. Inhibition of this enzyme results in the accumulation of acetylcholine in the nerve synapses, resulting in disrupted nerve function. Chronic inhibition of 50 percent of brain AChE has been associated with mortality in birds (Ludke $et\ al$. 1975; Hill and Fleming 1982).

The effects of DDTr on other receptor groups are not as clearly defined as in birds. Recent studies indicate that DDTr (especially o,p' isomers) may mimic estrogen, resulting in adverse reproductive effects. Observed effects include feminization and increased female:male population ratios for some receptors. Other responses include histopathological changes, alterations in thyroid function, and changes in the activity of various enzyme groups (Peakall 1993).

E.3.2   Dieldrin

Dieldrin is a non-systemic and persistent cyclodiene insecticide. It was broadly used in the United States until 1974, when the EPA restricted its use to termite control via direct soil injection and for non-food seed and plant treatment. Dieldrin is no longer produced commercially in the U.S. (HSDB 1997).

Dieldrin is extremely persistent in the environment due to its extremely low volatility and low solubility in water. The time required to degrade 95% of dieldrin in soil has been estimated to vary from 5 to 25 years. It is highly lipophilic and is therefore prone to bioaccumulate and biomagnify (HSDB 1997). A variety of bioconcentration factors have been calculated for dieldrin, ranging from

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128 for an alga to 68,286 for whole body yearling lake trout (U.S. EPA 1980).

In the aquatic environment, dieldrin is extremely toxic, with 96-hour acute LC50's ranging from 5.0 ug/L for the isopod *Asellus brevicaudus* to 740 ug/L for a crayfish. For fish, the most sensitive species is the rainbow trout, with a 96-hour LC50 of between 1.1 and 9.9 ug/L. The most resistant fish species is the goldfish, with a 96-hour LC50 of 41 ug/L. In a chronic study using *Daphnia magna*, a chronic value of 57 ug/L was obtained. Two chronic studies have been conducted using fish. One was an early-life stage exposure using rainbow trout in which a chronic value of 0.22 ug/L was obtained. The other study was a three-generation study using the guppy, in which a chronic value of 0.45 ug/L was obtained (U.S. EPA 1980).

In mammals, dieldrin is rapidly absorbed from the GI tract upon ingestion. It is then transported from the liver to various tissues in the body, including the brain, blood, liver, and adipose tissue. Dieldrin is metabolized by the mixed function oxidase (MFO) enzyme system. For most species (rat, mouse, dog, monkey, and sheep), the acute oral toxicity is between 20 and 70 mg/kg. The toxicity appears to be related to the central nervous system, with stimulation, hyperexcitability, hyperactivity, incoordination, and exaggerated body movement, ultimately leading to confusion, depression, and death (EPA 1980).

Dieldrin has been shown to cross the placental barrier, and for that reason has been studied for its teratogenic properties and reproductive effects. When mice were fed 25 mg/kg of dieldrin in the diet for six generations, parameters such as fertility, gestation, viability, lactation, and survival of the young were adversely affected. When hamsters were fed one dose equivalent to one-half the LD50 of dieldrin, increased fetal death, decreased fetal growth, open eye, webbed feet, cleft palate, and other effects were observed. Two later studies were performed in which lower dosages of dieldrin were administered, and equivocal results were obtained (U.S. EPA 1980).

In birds, the oral LD50 of dieldrin was determined to be 6.9 mg/kg BW using the sharp-tailed grouse. A variety of reproductive effects have also been observed in birds, including decreased egg production and fertility. Studies have shown that organochlorine insecticides induce liver enzymes that lower estrogen levels and result in late breeding and other related reproductive manifestations. A correlation has also been established between egg concentrations of dieldrin, eggshell thickness, and hatching success. In addition, studies in male chickens, pheasants and quail have indicated that dieldrin causes a reduction in testicular size and alters homone metabolism (U.S. EPA 1976).

E.3.3   Heptachlor

Heptachlor is used in the United States primarily for termite and other wood-destroying insect control (in 1983, the use of heptachlor as an insecticide was restricted to termite control). The application of this pesticide thus results in the contamination of soil surrounding wooden structures and buried cable closures (U.S. EPA 1988). The release of heptachlor to soil surfaces results in the volatilization of this compound, usually occurring more rapidly in moist soil than in dry soil. Hydrolysis is expected to be a significant mechanism of heptachlor removal if the compound is incorporated into the soil. The half-life of heptachlor in soil was calculated to range from 0.4 to 0.8 years. In soil, heptachlor will degrade to 1-hydroxychlordene, heptachlor epoxide, and an unidentified metabolite that is hydrophilic than heptachlor epoxide. Biodegradation may be important, especially under anaerobic conditions. Heptachlor is expected to adsorb strongly to soils and, therefore, to resist leaching into ground water. The release of heptachlor to water will result in hydrolysis to 1-hydroxychlordene (half-life of about 1 day) and volatilization. Bioconcentration by aquatic organisms may contribute to heptachlor removal from water. Bioconcentration factors for aquatic organisms and heptachlor are high; 3,800 in mosquito fish; 21,379 in sheepshead minnows; and 19,952 in fathead minnows. Bioconcentration may be limited, however, by the rapidity of heptachlor hydrolysis to 1-hydroxychlordene and the adsorption of heptachlor to sediments. The adsorption to sediments is expected to be slow compared

to hydrolysis (Howard 1991; U.S. EPA 1988).

Heptachlor exhibits moderate acute toxicity to terrestrial organisms but is extremely toxic to aquatic organisms and birds. It is persistent in the environment, virtually insoluble in water, stable in daylight and air, and bioaccumulates. In biological systems, heptachlor is rapidly epoxidized to heptachlor epoxide. The precise mode of action in biological systems is not known (U.S. EPA 1988). The toxic effects of heptachlor are not specific for any one organ system. The liver and central nervous system are most significantly affected by heptachlor. Effects have also been observed in the reproductive, hematopoietic, immune, and renal systems.

E.3.4   Methoxychlor

Methoxychlor is an insecticide that is structurally related to DDT in that the two chlorine atoms in the *para* position of the DDT molecule are replaced by a methoxy moiety in methoxychlor. Methoxychlor is less stable than DDT and thus has less residual effect than DDT (Matsumura 1975). It has been used on many agricultural crops and farm animals, and for spraying of barns, grain bins, mushroom houses, and other agricultural premises for the control of flies (HSDB 1997).

If released to soil, methoxychlor is expected to remain immobilized in the upper layer, although a small portion may migrate to lower depths, as suggested by detection of methoxychlor in some groundwater samples. Methoxychlor is relatively persistent in soil, and has been demonstrated to remain in soil for up to 14 months. In the aquatic environment, methoxychlor may be transported by adsorbing to suspended sediment particles. In sediments, it was found to have a half-life of > 100 days under aerobic conditions and < 28 days under anaerobic conditions. This, as well as other studies, indicate that methoxychlor will biodegrade rapidly under anaerobic conditions, but is not expected to biodegrade significantly under aerobic conditions. It may also undergo sensitized (indirect) photolysis, as well as limited chemical hydrolysis under moist conditions, and volatilization may also be an important removal pathway (HSDB 1997).

The major metabolic pathway for methoxychlor has been shown to be o-demethylation and subsequent conjugation, indicating that methoxychlor is metabolized by the mixed function oxidase (MFO) enzyme system. It has been estimated in rats that methoxychlor is stored in fat at only 0.01 to 0.1 times its chronic intake (HSDB 1997). Since methoxychlor is rapidly metabolized in the mammalian body and is not accumulated in fat or excreted in milk, it was preferred to DDT for use on animals, in animal feed, and in barns (Matsumura 1975). Bioconcentration Factor (BCF) values have been obtained for a variety of aquatic organisms, indicating that methoxychlor will bioaccumulate in aquatic organisms which do not metabolize methoxychlor at a significant rate, and that fish generally metabolize this compound very rapidly and thus will not tend to bioaccumulate it in their tissues (HSDB 1997).

In the aquatic environment, the isopod *Aselus communis* was found to be the most sensitive organism, with a 28-day LC50 of 0.42 ng/L. In seven species of aquatic invertebrates, the 96-hour LC50s ranged from 0.42 - 12 ug/L. Similar to DDT, the toxicity of methoxychlor to fish has been shown to vary with temperature in some species, with its toxicity increasing as temperature decreases. For example, at 12.7 °C, the 96-hour LC50s for rainbow trout and bluegill have been shown to be 62 and 75 ug/L, respectively, whereas at 1.6 °C, the LC50s were 30 and 42 ug/L, respectively. Overall, the acute toxicity of methoxychlor in fish is highly variable, with 96-hour LC50s for twenty-one species of fish ranging from 1.7 ug/L for the Atlantic salmon to 150 ug/L for the northern puffer (HSDB 1997).

In mammals, methoxychlor is only 1/25 to 1/50 as toxic as DDT. It has an oral LD50 of 6000 mg/kg in rats, reflecting its extremely low acute mammalian toxicity (Matsumura 1975). Methoxychlor affects the central nervous system and has also been shown to be estrogenic in vivo in the female. Its metabolite, the di-demethylated derivative of methoxychlor, has been shown to inhibit the binding of

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17-β-estradiol to rat uterine cytosolic estrogen receptor in vitro. Developmental and reproductive effects which have been observed as a result of exposure to methoxychlor include testicular atrophy, arrested spermatogenesis and folliculogenesis, reduced fertility, and wavy ribs (HSDB 1997).

Mallard ducks, sharp-tailed grouse, and California quail ingesting a median lethal dose of methoxychlor exhibited leg weakness (in mallards), jitterinesss, low stance, and wing spread, which disappeared within a few days. The oral LD50 has been calculated to be > 2000 mg/kg in the mallard duck, the sharp-tailed grouse, and the Califorina quail (HSDB 1997).

E.3.5   Endrin/Endrin Aldehyde/Endrin Ketone

Endrin was used as an insecticide, avicide, and rodenticide. Its general toxic effects include ataxia, slowness, drowsiness, tremors, trachael congestion, prostration, convulsions, wing-beat convulsions, and opisthotonos. Formulations of endrin generally contain impurities of related compounds, including endrin aldehyde and endrin ketone. These two chemicals are also known to be metabolites of the parent endrin compound. Since these two chemicals were detected in some of the samples collected at the Cornell Dubilier site, all three chemicals were evaluated together for the purposes of this risk assessment. Much of the information contained in this section, however, relates to endrin itself because most of the information in the literature revolves around the parent compound (HSDB 1997).

When endrin is released into the soil, it is not expected to migrate into the groundwater due to its expected strong adherence to soil particles. However, the detection of small amounts of endrin in some samples of groundwater indicate that some migration is possible. Endrin will persist in soil for long periods of time (up to 14 years or more). Small amounts of endrin may volatilize, and it has been shown to photodegrade to endrin ketone. However, biodegradation and hydrolysis are not important removal mechanisms. When endrin enters aquatic systems, it is expected to adsorb strongly to sediments, thus providing a potential aquatic transport mechanism, and evaporation from water is not expected to be significant. Endrin aldehyde and endrin ketone are expected to have a very similar fate in the environment as endrin (HSDB 1997).

The toxic mechanism of endrin is believed to include inhibition of the brain-specific (35)S-t-butylbicyclophosphorothionate binding site. It has also been shown that endrin produces specific alterations in unmyelinated fiber bundles of peripheral nerves but does not affect myelinated fibers. A variety of metabolites of endrin have been identified, including endrin ketone (12-ketoendrin) and endrin aldehyde, as mentioned previously. Additional metabolites that are believed to be significant include 9-ketoendrin, 9-hydroxyendrin, 3-hydroxyendrin, and trans-4,5-dihydroisodirn-4,5-diol. A variety of bioconcentration factors (BCFs) have been calculated, ranging from 140 in algae after seven days to 49,000 in a species of snail (Physa). In fish, the BCF has been calculated for a variety of species and ranged from 1335-10,000 (HSDB 1997).

Endrin has been shown to be extremely toxic to aquatic organisms. The toxicity of endrin has been tested in two species of *Daphnia*, resulting in 48-hour EC50s of 4.2 and 20 ug/L for *Daphnia magna* and *Daphnia pulex*, respectively. In addition, 96-hour LC50s for twelve species of benthic macroinvertebrates ranged from 0.08 to 62 ug/L. In eleven species of fish, the 96-hour LC50s ranged from 0.033 ug/L in *Ophiocephalus punctatus* to 1.8 ug/L in the fathead minnow (HSDB 997).

In mammals, the acute toxicity of endrin has been tested in a variety of species, and the oral LD50s ranged from 1.3 mg/kg in mice to 36 mg/kg in male Guinea pigs. No-effect levels have also been established, and are reported to be 0.05 mg/kg/day in the rat, 0.038 mg/kg/day in the mouse, and 0.025 mg/kg/day in the dog. Equivocal results have been obtained regarding the teratogenicity of endrin. For example, in one study in which one-half the LD50 was administered to pregnant hamsters and mice, increased fetal deaths, open eye, webbed foot, cleft palate, and fused ribs were observed in

young hamsters, but not in young mice. Other studies have indicated that dietary levels that do not injure the parents do not adversely affect development of the offspring. Endrin aldehyde is slightly less toxic than endrin, with an oral LD50 of 10 mg/kg in rats and 62 mg/kg in mice. Endrin ketone, however, has been shown to be more toxic than endrin, with an oral LD50 of 1.1 and 0.8 mg/kg in male and female rats, respectively. This indicates that endrin ketone may be responsible for much of the acute toxicity observed in mammals as a result of exposure to endrin (HSDB 1997).

In birds, the acute oral LD50s have ranged from 1.19 mg/kg in female California quail to 5.64 mg/kg in female mallard ducks. In the diet of poultry, 20 ppm of endrin produced anorexia, ataxia, convulsions, and death. The 30-day empirical minimum lethal dose for mallards has been calculated to be 0.25 mg/kg/day for both sexes. The reproductive and developmental effects of endrin in birds, as in mammals, have also been shown to be equivocal. For example, reduced egg production in quail and pheasants as well as reduced chick survival in pheasants have been observed as a result of exposure to endrin, but no reproductive effects were observed at similar concentrations in mallard ducks (HSDB 1997).

E.3.6   Chlordane

Technical chlordane is an organochloride pesticide that was introduced in the U.S. in 1947. (Eisler 1990). After concerns of its potential carcinogenicity, the production of chlordane was reduced and it was banned from use in the U.S. in 1983, except when used for the control of underground termites.

Technical chlordane consists of roughly 45 components, primarily cis-chlordane, trans-chlordane, heptachlor and various other chlordane isomers (Eisler 1990). The half-life of cis-chlordane in water is relatively short , between 1.1 and 17.5 h (Feroz and Khan 1979). However, in soil the half-life is much longer ranging anywhere from .5 to 10 years. Chlordane persists in soil because of its low solubility in water, relatively low vapor pressure and great tendency to adsorb to soil particles.

The major route of global dissemination for chlordane is generally considered to be atmospheric transport. Some chlordane isomers will persist in soil for 3 to 15 years but plants generally do not appear to accumulate it in their tissues. Chlordane concentrations in living organisms are typically greatest near areas where chlordane has been applied for termite and pest control, in predatory species and in tissues with high lipid content (Eisler 1990). Except in certain marine mammals, food chain biomagnification of chlordane is usually low (Eisler 1990)

Chlordane is a nerve stimulant causing lack of coordination and hyperexcitability in animals at low chronic doses and tremors and convulsions at high acute doses (Ingle 1965; Klassen et al. 1986). Chlordane is readily absorbed by warm-blooded animals through skin, diet and inhalation (Eisler 1990). Quickly distributed through the body, it concentrates in the liver and fat (WHO 1984). Large amounts of chlordane (75%) were absorbed in the gut in oral dosing studies using rats and mice (Nomeir and Hajjar 1987), while rabbits absorbed 33% after an oral dose (EPA 1988). Chlordane residues in mammals were not measurable 4 to 8 weeks after exposure (Ingle 1965).

Chlordane has been extensively applied to soil to control soil invertebrate pests. When applied at rates ranging from 0.6 and 2.24 kg/hr, non-target species such as earthworms were adversely affected (Eisler 1990). Bird species sensitive to chlordane had reduced survival after a single oral dose of 14.1 mg/kg BW/day. Aquatic species are also adversely affected at concentrations in water of 0.2 to 3.0 ug/L technical chlordane. The greatest concern over the use of chlordane is driven by findings of liver cancer in domestic mice after exposure to chlordane. In addition, mammals fed 0.76 to 5.0 mg/kg of chlordane in feed were found to have elevated tissue residues and growth inhibition. Once metabolized in mammals, chlordane primarily becomes oxychlordane which is 20 times more toxic than chlordane itself and persists in adipose tissue.

E.4   PCBs

A variety of PCB-induced toxic effects have been observed in mammals. Mink are particularly sensitive to dietary PCB levels (Aulerich et al. 1985). Anorexia, weight loss, lethargy, enlarged livers, and intestinal discharge of blood have been noted in exposed mink (Eisler 1986b). Placental and mammary transfer of PCB has been shown to be a direct route of PCBs between mother and young. PCB exposure can lead to behavioral disorders, specifically in sleep/wake cycles, and in animals that hibernate or aestivate (Montz et al. 1982; Sanders and Kirkpatrick 1977). Negative effects of PCBs on metabolism, thyroid control, ATPase activity, oxidative phosphorylation, steroid hormone activity, immunity, and vitamin A pathways have been noted (Safe 1984; U.S.EPA 1980a).

PCB toxicity in mammals is highly variable. While some PCBs are extremely toxic, and can produce death and cause reproductive failure in very low levels, others appear to produce few, if any, toxic responses (Eisler 1986b). Toxic responses to PCBs are highly species specific. Mink are highly susceptible to PCB toxicity, while closely related mammals, such as the European ferret, are more resistant (Eisler 1986b). Younger mammals appear to be more susceptible to PCB poisoning than adults (Eisler 1986b) Mutagenic, carcinogenic, and teratogenic effects of PCB exposure have been observed, with mutagenic activity appearing to increase with increasing chlorination of the PCB molecule (Eisler 1986b).

As with mammals, there is also a great degree of variability among different bird species in response to PCBs. In sensitive species, normal patterns of growth, behavior, reproduction, and metabolism may be altered. Liver concentrations of PCBs are generally highest in piscivorous birds, followed by birds that feed on other smalls bird and mammals, birds that feed on worms and insects, and herbivorous or seed eating birds, respectively (NAS 1979).

E.5   Metals

E.5.1   Aluminum

Because of its strong reactivity, aluminum (Al) is not found as a free metal in nature. Aluminum has only one oxidation state (+3), thus its behavior in the environment depends on its ordination chemistry and the surrounding conditions. In soils, a low pH generally results in an increase in aluminum mobility. In water, an equilibrium with a solid phase is established that controls the extent of aluminum dissolution (ATSDR 1990a).

Plants vary in their ability to remove aluminum from soils, although bioconcentration factors for plants are generally less than one. Biomagnification of aluminum in terrestrial food chains does not appear to occur. There is no data on the biomagnification of aluminum in aquatic food chains (ATSDR 1990a).

The nervous system may be a target area for aluminum. Aluminum accumulates in neurofibrillary tangles in humans with Alzheimer's disease. Aluminum may also interact with neuronal DNA to alter gene expression and protein formation. Mammalian studies do not indicate that aluminum affects reproduction although some developmental effects have been reported in mammals (ATSDR 1990a).

E.5.2   Antimony

Antimony (Sb) is a silvery white metal of medium hardness and low solubility in water. It is found at very low levels in the environment. Metallic antimony is stable under ordinary conditions and is not readily altered by air or water. Antimony displays four oxidation states, Sb(-3), Sb(0), Sb(+3), and Sb(+5). The +3 state is the most common and stable (ATSDR 1991).

The speciation and physicochemical state of antimony are important to its behavior in the environment and availability to biota. Antimony that is incorporated into mineral lattices is inert and unlikely to be bioavailable. Unfortunately, most analytical methods for antimony do not distinguish between this form and adsorbed forms. Little is known about the adsorption of antimony in soil; however, since antimony forms anionic species, adsorption should be greatest under weakly acidic conditions. Antimony's adsorption to soil and sediment is primarily correlated with the iron, manganese, and aluminum content; it coprecipitates with hydroxylated oxides of these elements (ATSDR 1991).

As a natural constituent of soil, antimony is transported into stream and waterways from natural weathering of soil and anthropogenic sources. Antimony has a low occurrence in ambient waters. Antimony in aerobic freshwater and seawater is largely in the +5 oxidation state. Trivalent antimony is the dominant oxidation state in anaerobic water. Antimony can be reduced and methylated by microorganisms in anaerobic sediment, releasing volatile methylated antimony compounds into the water (ATSDR 1991).

Antimony does not appear to bioconcentrate appreciably in fish or other aquatic organisms. Much of the antimony occurring in plants has been found to be a result of surface deposition. Uptake of antimony from soil by plants is reported to be minor. Body burden analyses of terrestrial organisms suggest that biomagnification of antimony does not occur from lower to higher trophic levels (ATSDR 1991).

The majority of effects resulting from the inhalation of antimony is attributed to the accumulation of antimony dust in the lung (pneumoconiosis) which may progress to a proliferation of alveolar macrophages to fibrosis. The heart is another target organ in antimony exposure, resulting in altered blood pressure, increased heart rate, and decreased contractile force. Antimony is known historically for its emetic properties, causing vomiting, diarrhea, gastric discomfort, and ulcers. Dietary exposure studies have reported decreased hemoglobin and hematocrit levels, altered erythrocyte counts, and swelling of the hepatic cords (ATSDR 1991).

E.5.3   Arsenic

Arsenic has four valence states (-3, 0, +3, and +5), rarely occurring in its free state in nature. It is usually a component of sulfidic ores, occurring as arsenides and arsenates, along with arsenic trioxide, which is a weathering product of arsenides. Biotransformations may occur, resulting in volatile arsenicals that normally are returned to land where soil adsorption, plant uptake, erosion, leaching, reduction to arsines, and other processes occur. Inorganic arsenic is more mobile than organic arsenic, and thus poses greater problems by leaching into surface waters and groundwater. The trivalent arsenic species (+3) are generally considered to be more toxic, more soluble, and more mobile than As (+5) species (Eisler 1988a).

Arsenic in water exists primarily as a dissolved ionic species. Particulates account for less than one percent of the total measurable arsenic. Arsenates are more strongly adsorbed to sediments than are other arsenic forms. In bodies of water that become stratified in summer, arsenic released from sediments accumulates in the hypolimnion until turnover, when it is mixed with epilimnetic waters. This mixing may result in a ten to twenty percent increase in arsenic concentrations (Eisler 1988a).

Eisler (1988a) reports the following points to be agreed upon by most investigators: (1) arsenic may be absorbed by ingestion, inhalation, or permeation of the skin or mucous membrane, (2) cells accumulate arsenic by using an active transport system normally used in phosphate transport, (3) arsenicals are readily absorbed after ingestion, most being rapidly excreted in the urine during the first few days, (4) the toxicity of arsenicals conforms to the following order from greatest to least toxicity: arsines > inorganic arsenites > organic trivalent compounds (arsenoxides) > inorganic arsenates > organic pentavalent compounds > arsonium compounds > elemental arsenic, (5) solubility in water

and body fluids appear to be directly related to toxicity, and (6) the mechanisms of arsenical toxicity differ considerably among arsenic species, although signs of poisoning appear similar for all arsenicals.

The primary mechanism of inorganic trivalent arsenic toxicity is through reaction with sulfhydryl groups of proteins and subsequent enzyme inhibition; inorganic pentavalent arsenic does not react as readily with sulfhydryl groups. Inorganic trivalent arsenic interrupts oxidative metabolic pathways and sometimes causes morphological changes in liver mitochondria. Methylation greatly reduces the toxicity of inorganic arsenic (both trivalent and pentavalent) and is usually the major detoxification mechanism (Eisler 1988a).

The mechanism of organic arsenic toxicity begins with its initial metabolism to the trivalent arsenoxide form, followed by its subsequent reaction with sulfhydryl groups of tissue proteins and enzymes, to form an arylbis (organylthio) arsine. This form inhibits oxidative degradation of carbohydrates and decreases cellular ATP (Eisler 1988a).

E.5.4   Barium

Because barium is an element, it does not degrade. Barium is widely distributed in both terrestrial and aquatic environments. Based on its Kd (60 in Baes et al. [1984]), barium would be expected to adhere to particulate matter. Although barium is found in most aquatic environments, most barium precipitates out in the form of insoluable salts (EPA 1986). Transport of barium by suspended sediments in lotic water bodies may be significant. Based on its Henry's Law Constant (value of zero in EPA [1992]), volatilization should not be a significant fate process. Barium is not expected to bioconcentrate significantly in plants or freshwater aquatic organisms.

Barium occurs naturally in most surface water and groundwater. In groundwater and surface water, barium is likely to precipitate out of solution as an insoluable salt (EPA 1986). The chemical form of barium largely dictates its adsorption into soils and sediments. Barium in sediments is found largely in the relatively insoluble form of barium sulfate and also in the insoluable form of barium carbonate. Humid and fulvic acid have not been found to increase the mobility of barium (ATSDR 1990c). Based on its Henry's Law Constant (value of zero in EPA [1992]), volatilization from surface water should not be a significant fate process.

Based on its Kd (value of 60 reported in Baes et al. [1984]), barium would be expected to adsorb to soil and sediment. Soils with high cation exchange capacity adsorb barium and limit its mobility. Barium is more mobile and more likely to be leached from soils in the presence of chloride due to the solubility of barium chloride relative to other forms of barium (ATSDR 1990c).

Barium will be taken up by plants under certain environmental conditions, but generally at concentrations less than the surrounding soils (Baes et al. 1984). While bioconcentration has been found to be significant in marine systems, it is less significant in freshwater systems (ATSDR 1990c).

E.5.5   Beryllium

The majority of the beryllium (Be) in the environment is the result of coal and oil combustion. Beryllium naturally enters waterways through the weathering of rock and soil, and through deposition of atmospheric beryllium. Upon reaching water and soil, beryllium is most likely retained as an insoluble form that is generally immobile. However, beryllium chloride, fluoride, nitrate, phosphate, and sulfate (tetrahydrate) are all water-soluble forms. Although chemical reactions may transform one beryllium compound into another, beryllium cannot be degraded by environmental reactions (ATSDR 1993a).

Due to its geochemical similarity to aluminum, beryllium may be expected to adsorb onto clay surfaces at low pHs, and it may remain precipitated as insoluble complexes at higher pHs. Therefore, beryllium is expected to have limited mobility in soil (ATSDR 1993a).

Beryllium is not expected to bioconcentrate in aquatic animals and no evidence for significant biomagnification within food chains has been found. Beryllium is extremely toxic to warmwater fish in soft water. The degree of toxicity decreases with increasing hardness (ATSDR 1993a).

Major exposure routes for aquatic ecological receptors include ingestion of contaminated soil and sediment. Although several studies point out the negative effects of beryllium in mammalian systems, no studies that evaluated the relationship between sediment beryllium concentration and observed toxicity to benthic organisms could be found (ATSDR 1993a).

E.5.6 Cadmium

Cadmium (Cd) is a mutagen, teratogen, and a suspected carcinogen (RTECS 1997). Tissue levels of cadmium increase with the age of an organism and eventually act as a cumulative poison (Hammons et al. 1978). Cadmium replaces essential metals (e.g., zinc) at critical sites on proteins and enzymes, and may inhibit a variety of enzymatic reactions. It inhibits Phase I and Phase II biotransformation reactions, probably by alteration of the enzymes responsible for these reactions (Sipes and Gandolfi 1986). Cytochrome P-450 monoxygenases play a major role in Phase I reactions. Cadmium also combines with sulfhydryl groups in enzymes, which affects the transfer of electrons from compounds in the citric acid cycle to compounds in the electron transport chain. Cadmium can inhibit adenosine triphosphate (ATP) activity in the following ways: 1) it binds to and inactivates enzymes which synthesize ATP, and 2) it binds to ATPase, which is required to convert ATP to $ADP + PO_4$ (Hammons et al. 1978).

Vertebrates tend to accumulate cadmium in the kidney and liver tissue (Eisler 1985a). Freshwater aquatic species are most sensitive to toxic effects of cadmium, followed by marine organisms, birds, and mammals.

E.5.7 Calcium

Calcium (Ca) is the fifth most abundant element and the third most abundant metal on earth. It is widespread in nature as calcium carbonate (limestone and marbel), calcium sulfate, calcium fluoride (fluorspar) and calcium phosphate (apatite). It occurs in the earth's crust at a concentration of 3.65%, and in sea water at approximately 400 g/ton. It is an important constituent of bones, teeth, and shells of living organisms, and is essential for muscular, nervous system, and renal function, as well as for blood coagulation and respiration. Therefore, calcium is regulated by the body. In addition to its nutritional value, calcium is used in industry as a catalyst for polyester fibers, in metallury as a deoxidizer for copper, beryllium, and steel, as an alloying agent for aluminum, copper, and lead, and in many other uses (HSDB 1997).

The environmental toxicology of calcium has not been extensively evaluated due to its natural existence in the environment at high concentrations. However, it is reported to be dangerous to aquatic life in high concentrations (CHRIS 1997). In invertebrates, mechanisms to control the intracellular concentration of calcium are relatively well understood. One such mechanism is via precipitation of excess calcium into granules. It has been shown that Mn entering a cell can lead to the corrosion of the granule surface and the liberation of excess calcium into the cell, resulting in toxic effects, including necrosis of the hepatopancreas (Newman and McIntosh 1991).

In mammals, it has been shown that chronic ingestion of calcium carbonate may cause hypercalcemia, alkalosis, and renal failure. Hypercalcemia may affect the heart and the nervous system. Neurologic effecst might include confusion, coma, decreased deep tendon reflexes, depression, fatigue, hallucinations, lethargy, or weakness (Meditext 1997).

E.5.8 Chromium

Chromium (Cr) can exist in oxidation states ranging from -2 to +6, but it is most frequently converted to the relatively stable trivalent (+3) and hexavalent (+6) oxidation states (Eisler 1986b). In both freshwater and marine systems, hydrolysis and precipitation are the most important processes that determine the fate and effects of chromium, whereas adsorption and bioaccumulation are relatively minor. Precipitated Cr^{+3} hydroxides remain in sediments under aerobic conditions. However, under anoxic and low pH conditions, Cr^{+3} hydroxides may solubilize and remain as ionic Cr^{+3} unless oxidized to Cr^{+6} through mixing and aeration (Eisler 1986b). In soils, the solubility and bioavailability of chromium are governed by soil pH and organic complexing substances, although organic complexes play a more significant role (James and Bartlett 1983a, James and Bartlett 1983b).

The trivalent state is the form usually found in biological materials. This form functions as an essential element in mammals by maintaining efficient glucose, lipid, and protein metabolism (Steven et al. 1976). Chromium is beneficial but not essential to higher plants (Eisler 1986b). The biomagnification and toxicity of Cr^{+3} is low relative to Cr^{+6} because of its low membrane permeability and its noncorrosivity. However, a large degree of accumulation by aquatic and terrestrial plants and animals in the lower trophic levels has been documented (Eisler 1986b), although, the mechanism of accumulation remains largely unknown.

Chromium is mutagenic, carcinogenic, and teratogenic, with Cr^{+6} exhibiting the greatest toxicity; relatively less is known about the toxicity of Cr^{+3}. At high concentrations, Cr^{+6} is associated with abnormal enzyme activity, altered blood chemistry, lowered resistance to pathogenic organisms, behavioral modifications, disrupted feeding, histopathology, osmoregulatory upset, alterations in population structure, and inhibition of photosynthesis (Eisler 1986b).

Rabbits fed dietary chromium accumulated hyaluronates, chondroitin sulfates, and neutral mucopolysaccharides in the soft tissues, causing pericapillary sclerosis (Kucher and Shabanov 1967). This accumulation blocked blood tissue barriers which are normally permeable, preventing the normal transport of metabolites. One manifestation of this condition was the inhibition of insulin production in the pancreatic islets due to damage to the beta-cells contained therein.

Chromium also leads to nephron damage via swelling and loss of microvilli, the formation of intracellular vacuoles, mitochondrial swelling, and cytoplasmic liquefication and loss of cells lining the nephron surface (Evan and Dail 1974).

The preliminary step in chromium-induced respiratory cancer is speculated to be the scarring of alveolar tissue, followed by the elicitation of inflammatory reactions in lung tissue leading to bronchopneumonia, alveolar epithelial changes, atrophy, and benign tumor formation. Direct skin contact with highly corrosive chromic acid and its anhydride produces skin ulcers and necrosis by a mechanism independent of any allergic response (Steven et al. 1976).

E.5.9 Cobalt

Because cobalt is an element, it does not degrade. Cobalt is widely distributed in nature and comprises 0.001-0.002% of the earth's crust. Based on its Kd (45 in Baes et al. [1984]), cobalt would be expected to adhere to particulate matter. Most cobalt in water is precipitated or adsorbed onto suspended soils and sediments. Cobalt may bioaccumulate in plants and aquatic organisms (ATSDR 1992a; HSDB 1997).

Cobalt is relatively insoluble in cold and hot water, but is soluable under acidic conditions (HSDB 1997). The speciation and subsequent transport of cobalt in water is affected by a number of factors including the presence of ligands, the concentration of anions, pH, and Eh. Depending on the nature

of the water, the amount of dissolved, suspended, and sedimented forms may vary substantially. Cobalt is not significantly adsorbed by organic materials (e.g. humic and fulvic materials) in water. Most cobalt in water is precipitated or adsorbed onto suspended solids and sediments (ATSDR 1992a).

Cobalt is usually found in soils in the divalent state. Based on its Kd (45 in Baes et al. [1984]), cobalt would be expected to adhere to particulate matter. The mobility of cobalt in soils is primarily regulated by pH, with increasing mobility as the pH decreases (HSDB 1997; ATSDR 1992a). The mobility of cobalt also decreases as the availability of oxides (such as iron and mangenese oxides), crystalline materials, and other adsorbents in soil decreases (ASTDR 1992a).

Cobalt will be taken up by plants, but generally at concentrations less than the surrounding soil (Baes et al. 1994). In highly acidic soils, significantly higher than normal concentrations of cobalt have been found in plants. The translocation of cobalt from roots to above-ground parts of plants is not significant in most soils. The bioaccumulation factors for cobalt in marine and freshwater fish are 100-4000 and 40-1000, respectively, indicating some potential for bioaccumulation (ATSDR 1992a).

E.5.10 Copper

Copper (Cu) does not appear to have mutagenic properties, but it is a teratogen (RTECS 1997) and a possible carcinogen (Venugopal and Luckey 1978). Copper is caustic, and acute toxicity is primarily related to this property (Hatch 1978). Copper is an essential element for animals and is a component of many metalloenzymes and respiratory pigments (Demayo et al. 1982). It is also essential for iron utilization and functions in enzymes for energy production, connective tissue formation, and pigmentation (Venugopal and Luckey 1978). Excess copper ingestion leads to accumulation in tissues, especially in the liver. High levels of copper modify hepatic metabolism (Brooks 1988), which may lead to inability of the liver to store and excrete additional copper. When the liver concentration exceeds a certain level, the metal is released into the blood, causing hemolysis and jaundice. High copper levels also inhibit essential metabolic enzymes (Demayo et al. 1982). Toxic symptoms appear when the liver accumulates 3 to 15 times the normal level of copper (Demayo et al. 1982).

Although the exact mechanism of copper toxicity is not known, the following mechanisms have been proposed: formation of stable inhibitory complexes with cytochrome P-450 (Wiebel et al. 1971); impairment of function of NADPH-cytochrome reductase and alteration of mixed function oxidations (Reiners et al. 1986); and inhibition of heme biosynthesis (Martell 1981). Intranuclear inclusions may act as a detoxifying mechanism where copper is complexed by protein ligands, protecting cytoplasmic organelles (Demayo et al. 1982).

Ruminants are the most sensitive mammal species to copper toxicosis. Young animals retain more dietary copper than older animals and are more sensitive to copper toxicity (Venugopal and Luckey 1978).

E.5.11 Iron

Iron (Fe) is commonly detected at concentrations of 5 percent or more in soil. It is used primarily in the production of steel and other alloys as well as a major source of hydrogen. Iron is a constituent of hemoglobin and is essential to plant and animal life as well as being an important component in cellular oxidative processes. The disposition of ingested iron is regulated by a complex mechanism to maintain homeostasis. Therefore, bioconcentration in biota is not expected to be a significant process for iron. Generally, about 2 to 15 percent of ingested iron is absorbed from the gastrointestinal tract, and elimination is approximately 0.01 percent of the body burden per day. Adverse effects of iron toxicity may include renal failure and hepatic cirrhosis. The mechanism of toxicity begins with acute mucosal cell damage and absorption of ferrous ions directly into circulation, resulting in capillary endothelial cell damage to the liver (Shacklette and Boerngen 1984).

E.5.12 Lead

Lead (Pb) does not biomagnify to a great extent in food chains, although accumulation by plants and animals has been extensively documented (Wixson and Davis 1993; Eisler 1988b). Older organisms typically contain the highest tissue lead concentrations, with the majority of the accumulation occurring in the bony tissue of vertebrates (Eisler 1988b).

Predicting the accumulation and toxicity of lead is difficult since its effects are influenced to a very large degree, relative to other metals, by interactions among physical, chemical, and biological variables. In general, organolead compounds are more toxic than inorganic lead compounds, and young, immature organisms are most susceptible to its effects (Eisler 1988b). In plants, lead inhibits growth by reducing photosynthetic activity, mitosis, and water absorption. The mechanism by which photosynthetic activity is reduced is attributed to the blocking of sulfhydryl groups, inhibiting the conversion of coproporphyrinogen to proporphyrinogen (Holl and Hampp 1975).

The toxic effects of lead on aquatic and terrestrial organisms are extremely varied and include mortality, reduced growth and reproductive output, blood chemistry alterations, lesions, and behavioral changes. However, many effects exhibit general trends in their toxic mechanism. Generally, lead inhibits the formation of heme, adversely affects blood chemistry, and accumulates at hematopoietic organs (Eisler 1988b). At high concentrations near levels causing mortality, marked changes to the central nervous system occur prior to death (Eisler 1988b).

Plants can uptake lead through surface deposition in rain, dust, and soil, or by uptake through the roots. The ability of a plant to uptake lead from soils is inversely related to soil pH and organic matter content. Lead can inhibit photosynthesis, plant growth, and water absorption.

E.5.13 Magnesium

Magnesium (Mg) does not exist in a pure state in nature but is generally found in one of the following forms: dolomite, magnesite, brucite, periclase, carnallite, and kiersite. It is present in the earth's crust at about 2.1% by weight and thus is one of the most common elements in the earth's crust. In addition, it is found as a silicate in asbesteos and talc and is widely distributed. Magnesium is used for a variety of purposes, including as a constituent in light alloys, in the manufacturing of precision instruments, in pyrotechnics, for flash bulbs and flares, for grignard reagents, in the recovery of titanium, as an antiknock additive in gasoline, in batteries, and in many other applications (HSDB 1997).

Magnesium is an essential nutrient at low doses, and is therefore highly regulated in organisms. However, magnesium can become toxic at very high doses. In rats, a diet containing 49 mmol Mg/kg was reported to be magnesium adequate, while 8 mmol Mg/kg was reported to be magnesium deficient. Hypermagnesemia may cause impairment of neuromuscular transmission and cardiac effects (HSDB 1997).

The aquatic toxicity of magnesium has not been largely studied, due to its natural presence at high concentrations in surface waters. However, it has been shown to cause lethality after 6 hours at a concentration of 400 mg/kg in water using the stickleback as the test organism, and a 48-hour TLM of 1500 mg/kg was obtained using a marine fish. It has also been reported to have the potential to smother benthic organisms. As for the terrestrial environment, an oral LCLo of 230 mg/kg BW was reported, and a concentration of 500 ppm was reported to be toxic to livestock (OHM/TADS 1997).

E.5.14 Manganese

Manganese (Mn) does not occur as a free metal in the environment but is a component of numerous minerals. Elemental manganese and inorganic manganese compounds have negligible vapor pressures, but may exist in air as suspended particulate matter derived from industrial emissions or

al. 1982, Elhassani 1983). Mercury also binds strongly with sulfhydryl groups. Phenyl- and methylmercury compounds are among the strongest known inhibitors of cell division (Birge et al. 1979). In mammals, methylmercury irreversibly destroys the neurons of the central nervous system.

For all organisms tested, early developmental stages were most sensitive to toxic effects of mercury. Organomercury compounds, especially methylmercury, were more toxic than inorganic forms. In aquatic organisms, mercury adversely affects reproduction, growth, behavior, osmoregulation and oxygen exchange. At comparatively low concentrations in birds and mammals, mercury adversely affects growth and development, behavior, motor coordination, vision, hearing, histology, and metabolism. In mammals, the fetus is the most sensitive life stage (Eisler 1987a).

E.5.16 Nickel

Pure nickel (Ni) is a hard, white metal that is usually used in the formation of alloys (such as stainless steel), and nickel combined with other elements is found in all soils. Nickel is the twenty-fourth most abundant element and is found in the environment as oxides or sulfides. It may be released into the environment through mining, oil-burning power plants, coal-burning power plants, and incinerators. Nickel will attach to soil or sediment particles, especially those containing iron or manganese. Under acidic conditions, nickel may become more mobile and seep into the groundwater. The typical nickel concentration reported in soils is from 4 - 80 mg/kg. The speciation and physicochemical state of nickel is important in considering its behavior in the environment and its availability to biota (ATSDR 1996).

The most probable exposure routes of nickel is through dermal contact, inhalation of dust, and ingestion of nickel-contaminated soil. The respiratory system is the primary target of nickel exposure following inhalation. Manifestations such as inflammation of the lungs, fibrosis, macrophage hyperplasia, and increased lung weight have been noted in animals exposed to nickel. Animals exposed to nickel through oral exposure were noted to have lethargy, ataxia, irregular breathing, salivation, and squinting (ATSDR 1996).

E.5.17 Potassium

Potassium (K) occurs in the earth's crust at a concentration of 2.59% by weight and in seawater at a concentration of 3.8×10^{-5} ug/L. With the exception of lithium, it is the lightest known metal and is one of the most reactive and electropositive of metals. It is used in the synthesis of inorganic potassium compounds, as a heat transfer medium together with sodium, as a compounent of fertilizer, as a laboratory reagent for organic synthesis, and in the seeding of combustion gases in magnetohydrodynamic generators (HSDB 1997).

Potassium is reported to be harmful to aquatic life at very low concentrations. A freshwater TLm of 80 mg/L was obtained in a 24-hour acute toxicity test using mosquito fish (CHRIS 1997).

No information about the terrestrial toxicity or the mechanism of potassium toxicity was available at the time of report completion.

E.5.18 Selenium

Selenium (Se) chemistry is complex, existing as six stable isotopes of varying allopatric forms and valence states. Of these isotopes, Se-80 and Se-78 are the most common. Soluble selenates (+6) occur in alkaline soil and are slowly reduced to selenites (+4) which are readily taken up by plants. In acid or neutral soils, the amount of biologically available selenium should steadily decline. Selenium volatilizes from soils at rates that are modified by temperature, moisture, time, season, concentration of water-soluble selenium, and microbiological activity (Eisler 1985c).

the erosion of soil. Removal from the atmosphere is mostly through gravitational settling. The transport and partitioning of manganese in water is controlled by the solubility of the specific chemical form present. The metal may exist in water in any of four oxidation states (2+, 3+, 4+, or 7+). Divalent manganese (Mn+2) predominates in most waters (pH 4 to 7), but may become oxidized at a pH greater than 8 or 9. Manganese is often transported in moving water as suspended sediments. The tendency of soluble manganese compounds to adsorb to soils and sediments depends mainly on the cation exchange capacity and the organic composition of the soil. Manganese in water may be significantly bioconcentrated at lower trophic levels. However, biomagnification in the food chain may not be significant (ATSDR 1990).

The amount of manganese absorbed across the gastrointestinal tract is variable. There does not appear to be a marked difference between manganese ingested in food or in water. One of the key determinants of absorption appears to be dietary iron intake, with low iron levels leading to increased manganese absorption. This is probably because both iron and manganese are absorbed by the same transport system in the gut (ATSDR 1990).

E.5.15 Mercury

Mercury (Hg) may be present in the environment in a number of forms. In all inorganic forms, Hg^{2+} is the toxic species. The most toxic and bioavailable form of mercury is methylmercury (MeHg), which is highly stable and lipophilic, accumulating in food chains. Mercury can become methylated biologically or chemically. Microbial methylation of mercury occurs most rapidly under anaerobic conditions, common in wetlands and aquatic sediments. The majority of mercury detected in biological tissues is present in the form of methylmercury (Huckabee et al. 1979).

Mercury has no known biological function, and its presence in biological systems appears to result in undesirable effects. A number of toxic responses have been reported for mercury exposure. Eisler (1987a) reports that juvenile life stages are most susceptible to acute effects of mercury exposure. In fish, acute exposure results in impaired respiration, sluggishness, and loss of equilibrium (Armstrong 1979).

Mercury is a potent neurotoxin, resulting in impaired muscular coordination, weight loss, and apathy in birds, mammals, and fish (Eisler 1987a). Other reported effects include histopathological changes, changes in enzyme activity levels, mutagenicity, teratogenicity, and reproductive impairment. Mercury, especially methylmercury, is known to concentrate in biological tissues and magnify through the food chain.

Mercury can exist in three oxidation states: elemental mercury (Hg^0), mercurous ion (Hg_2^{2+}), and mercuric ion (Hg^{2+}). The mercuric ion is the most toxic inorganic chemical form (Clarkson and Marsh 1982). Methylmercury is the most hazardous form of mercury due to its high stability, its lipid solubility, and the ability to penetrate membranes in living organisms (Beijer and Jernelov 1979).

Mercury and its compounds have no known biological function. It is a mutagen, teratogen, and carcinogen, and causes embryocidal, cytochemical, and histopathological effects. Forms of mercury with relatively low toxicity can be transformed into forms of very high toxicity, such as methylmercury, through biological processes. In addition, mercury can be bioconcentrated in organisms and biomagnified through food chains.

Mercury in soils is generally not available for uptake by plants, due to the high binding capacity to clays and other charged particles (Beauford et al. 1977). Mercury levels in plant tissues increase as soil levels increase, however 95 percent of the accumulation and retention of mercury is in the root system (Beauford et al. 1977, Cocking et al. 1991).

All mercury compounds interfere with thiol metabolism in organisms, causing inhibition or inactivation of proteins containing thiol ligands and ultimately leading to mitotic disturbances (Das et

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Concentrations of selenium in water are largely a function of selenium levels in drainage systems and of water pH. High selenium levels tend to be associated with high water pH. Selenates represent the dominant species in drinking water. In seawater, selenites are the dominant species under some conditions. Selenites are less soluble than selenates and are easily reduced to elemental selenium. Elemental selenium is insoluble and largely unavailable although it is capable of satisfying nutritional selenium requirements (Eisler 1985c).

Selenium is an essential nutrient for some plants and animals, constituting an integral part of the enzyme glutathione peroxidase and may have a role in other compounds such as vitamin E and the enzyme formic dehydrogenase. Selenium also forms part of certain proteins, including cytochrome C, hemoglobin, myoglobin, myosin, and various ribonucleoproteins. In many systems, selenium deficiency is a larger problem than selenium toxicity (Eisler 1985c).

Selenium accumulation in certain species of plants may be extremely high. Consumption of selenium-accumulating plants by livestock has induced illness and death. Plants that accumulate selenium tend to be more deep-rooted than grasses, thereby surviving aridity and remaining as the principal forage for herbivorous animals. Concentration of selenium in animals tends to be higher in older than in younger individuals. In livestock, selenium is distributed by the circulatory system to all body organs. Concentrations tend to be highest in the liver, blood, kidney, spleen, and brain, and lowest in muscle, skin, hair, and bone. Elimination is primarily by urine, and smaller amounts are excreted with feces, breath, perspiration, and bile (Eisler 1985c).

Sublethal effects of elevated levels of selenium in diet or water are associated with reproductive abnormalities, congenital malformations, selective bioaccumulation, growth retardation, chromosomal aberrations, intestinal lesions, shifts in community composition, and behavioral modifications (Eisler 1985c).

E.5.19   Silver

Silver (Ag) is a rare element, but occurs naturally in the environment. There are no man-made sources of silver. Silver is used to make jewelry, silverware, electronic equipment, and dental fillings. The photographic industry uses silver compounds to make photographs. Photographic materials are the primary source of silver release into the environment. Other sources include mining operations and the natural wearing down of silver-bearing rocks and soil by the wind and rain. Silver that is released into the environment may be carried long distances in air and water. Rain can wash silver compounds out of the soil and into the groundwater. Silver does remain stable in the environment in various forms. Silver does not break down and can change its form by combining with other substances.

Oral ingestion of silver appears to effect the ocular and neurological systems. Studies involving the ingestion of high levels of silver documented mortality and weight loss. Limited and inconclusive evidence was presented for effects on the reproductive and cardiovascular systems. No studies were presented for oral effects of silver on the respiratory, gastrointestinal, hematological, musculoskeletal, hepatic, or renal systems.

The extent of silver absorption through the oral ingestion route has been found to be associated with transit time through the gastrointestinal tract. The faster the transit time, the less silver is absorbed. The distribution of silver throughout the body depends upon the route and quantity of silver administered and its chemical form. Silver distributes widely in the body with high concentrations found in the liver, spleen, bone marrow, lymph nodes, skin and kidney. The deposition of silver in tissues is the result of the precipitation of insoluble silver salts, such as silver chloride and silver phosphate. These insoluble salts appear to be transformed into soluble silver sulfide albuminates, to bind to or form complexes with amino or carboxyl groups in RNA, DNA, and proteins, or to be reduced to metallic silver by ascorbic acid or catecholamines. Studies have not indicated that biomagnification of silver in the food chain is significant (HSDB 1997).

E.5.20 Sodium

Sodium (Na) is found in the earth's crust at approximately 2.83% by weight and is the principle cation in the hydrosphere. It does not exist in its free form in nature, but rather in its halide, silicate, or carbonate forms. Sodium is used in the manufacture of tetraethyl lead, in photoelectric cells, in organic syntheses, in sodium lamps, as a coolant in nuclear reactors. Sodium violently decomposes in water, forming sodium hydroxide and hydrogen (HSDB 1997).

Sodium is toxic to aquatic life only at high concentrations. A freshwater toxicity test indicated that 4720 mg/L was harmless to sticklebacks. In a 48-hour toxicity test using a saltwater species, a Tlm of 24000 - 25000 mg/L was obtained. Sodium is not expected to bioaccumulate in the food chain (OHM/TADS 1997).

No information about the terrestrial toxicity of sodium was available at the time of report completion.

E.5.21 Thallium

Thallium (Tl) is one of the most toxic of the heavy metals. It is distributed widely but it is generally present in very low concentrations. Metallic thallium is soft and malleable, similar to lead in both appearance and physical properties. Freshly-prepared thallium oxidizes rapidly. A hydroxide is formed in the presence of water. Inorganic thallium (I) compounds are more stable than the thallium (III) analogues in aqueous solution at neutral pH. In contrast, covalent organothallium compounds are stable only in the trivalent form (Mulkey and Oehme 1993).

Thallium is rapidly and completely absorbed by the respiratory system, gastrointestinal tract, or skin. Water-soluble fractions are distributed to the brain, heart, kidney, skeletal muscle, and testis. Thallium (+1) interferes with K+-dependent processes and mimics K+ in its movement and intracellular accumulation in mammals. The thallium cation is less rapidly released than the K+ cation once it moves into the cell. Thus, because of its large distribution volume and low free plasma concentration, renal excretion of thallium is slow. The kidneys filter thallium into the urine, the salivary glands and liver concentrate thallium in their secretions, and the intestinal mucosal cells actively transport thallium into the lumen of the gastrointestinal tract, where it can be reabsorbed or eliminated in the feces. A small amount is taken up and excreted in hair. A significant percentage of free plasma thallium crosses the placental barrier (Mulkey and Oehme 1993).

Thallium's ability to interfere with a variety of K+-dependent processes is thought to play a significant role in its toxicity. Thallium (+) and K+ are thought to have common cellular targets and receptor sites associated with biological activity and toxicity. Various K+-dependent proteins are known to possess a higher affinity for Tl+ than for K+, and Tl+ alters the activity of these enzymes and membrane transport proteins (Mulkey and Oehme 1993).

Thallium's calcophilic character may also contribute to its observed toxicity in animals. Thallium has a high affinity for natural ligands that contain sulfhydryl (-SH) groups. These groups are structurally important in several classes of enzymes (Mulkey and Oehme 1993).

Thallium has been shown to adversely affect protein synthesis. Mammalian ribosomes are strictly dependent on K+ and Mg+2 for normal interactions between ribosomal subunits. Thallium (+) can replace K+ causing progressive destabilization and irreversible damage to ribosomes. Interactions between thallium and riboflavin may play a role in toxicity. Thallium may impair cell energy metabolism by causing a deficiency of riboflavin and riboflavin-derived cofactors (Mulkey and Oehme 1993).

Thallium is teratogenic in chick embryos, causing achondoplasia, leg bone curvature, parrot-beak deformity, microcephaly, and decreased fetal size. Teratological investigations in mammals have

produced conflicting results (Mulkey and Oehme 1993).

E.5.22 Vanadium

Elemental vanadium does not occur naturally but it can exist in 50 different ores and fossil fuels. Other anthropogenic sources include acid-mine leachate, sewage sludge, and fertilizers. The principal use of vanadium is as an alloy constituent, especially in steel. The addition of vanadium to steel removes oxygen and nitrogen, which improves the strength. The average concentration of vanadium in the earths crust is 150 mg/kg and in the U.S. soils are 200 mg/kg (Byernum et al. 1974).

The release of vanadium to water and soil occurs as a result of the weathering of rocks and from soil erosion. This process usually converts the less-soluble trivalent form to the more-soluble pentavalent form. The mobility of vanadium in soil is affected by pH, redox potential, and the presence of particulates. Relative to other minerals, vanadium is mobile in neutral or alkaline soils and its mobility decreases in acidic soils (ATSDR 1991; Van Zinderen Bakker and Jaworski 1980).

In the terrestrial systems, bioconcentration is more common in lower plant species. In addition, vanadium concentrations in plants are dependent on the amount of water-soluble vanadium, pH, and growing conditions. Vanadium appears to be present in all terrestrial mammals but the concentrations are usually below the detection limits. The highest concentration of vanadium is usually found in the liver and skeletal tissues (ATSDR 1991).

Vanadium is very poorly absorbed into the gastrointestinal tract and the toxic mechanism of vanadium on the respiratory system is similar to other metals (Castronova et al. 1984). Vanadium damages the alveolar macrophages by decreasing the macrophage membrane integrity. Damaged macrophages inhibit the ability of the respiratory system to clear itself of other particles. In vitro experiments indicate that the mechanism of toxicity of vandium is by inhibiting sodium-potassium ATPase activity, which inhibits the sodium-potassium pump. This pump is necessary for the transport of material across cell membranes (Nechay and Saunders 1978).

E.5.23 Zinc

Zinc (Zn) is essential for normal growth and reproduction in plants and animals and is regulated by metallothioneins. Metallothioneins act as temporary zinc storage sites and aid in reducing the toxicity of zinc to both vertebrates and invertebrates (Olsson et al. 1989). Zinc is not known to bioaccumulate in food chains, because it is regulated by the body and excess zinc is eliminated.

Zinc has its primary metabolic effect on zinc-dependant enzymes that regulate the biosynthesis and catabolic rate of RNA and DNA. High levels of zinc induce copper deficiency and interfere with metabolism of calcium and iron (Goyer 1986). The pancreas and bone seem to be the primary targets of zinc toxicity in birds and mammals. Pancreatic effects include cytoplasmic vacuolation, cellular atrophy, and cell death (Lu and Combs 1988, Kazacos and Van Vleet 1989). Zinc preferentially accumulates in bone, and induces osteomalacia, a softening of bone caused by a deficiency of calcium, phosphorus and other minerals (Kaji et al. 1988). Gill epithelium is the primary target site in fish. Zinc toxicosis results in destruction of gill epithelium and tissue hypoxia (Spear 1981).

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APPENDIX F

Toxicity Profiles
Cornell-Dubilier Site
South Plainfield, NJ
April 1998

APPENDIX F

TOXICITY PROFILES

F.1   Chronic Toxicity Profiles for the Food Chain Model

F.1.1   a-Chlordane/g-Chlordane

F.1.1.1   Chronic a-Chlordane/g-Chlordane Toxicity to Birds

One study was found pertaining to the dietary toxicity of chlordane to the red-winged blackbird. A dietary 6-7 day LD50 of 150 mg/kg of technical chlordane was reported for the red-winged blackbird (Stickel et al. 1979). However, due to the short duration and high dose used in this study, dietary studies using other birds were used to assess the dietary toxicity of chlordane to the red-winged blackbird.

Two studies were found in which the dietary toxicity of chlordane to the mallard duck was examined. Hudson et al. (1984) reported an LD50 from a single oral dose of 1,200 mg/kg BW to 4- to 5-month old ducks. In another study, a 5-day LD50 of 858 mg/kg in diet was reported by Hill et al. (1975). However, due to the short duration and high doses used in these studies, dietary studies using other birds were used to assess the dietary toxicity of chlordane to the mallard duck.

No studies were found in which the dietary toxicity of chlordane to the green heron was examined. Therefore, dietary studies using other birds were used to assess the dietary toxicity of chlordane to the green heron.

In one study, common barn owls (*Tyto alba*) were fed diets containing 75 mg/kg until 50% died and the survivors were sacrificed and measured for residues. Mortality was reached on day 40 (Eisler, 1990). In a longer term study, European starlings (*Sturnus vulgaris*) were fed a diet of various chlordane concentrations of which the lowest concentration (0.19 mg/kg BW/day) caused 50% mortality in 57 days.

For this risk assessment, a dietary exposure level of 0.19 mg/kg BW/day was used as a LOAEL and a dietary exposure level of 0.019 mg/kg BW/day was used as a NOAEL to estimate the risk of chlordane to the selected avian receptors.

F.1.1.2   Chronic a-Chlordane/g-Chlordane Toxicity to Mammals

No literature was found pertaining to the dietary toxicity of chlordane to the raccoon or the red fox. Therefore, literature pertaining to the dietary toxicity of chlordane to other mammals was reviewed. In one study, an acute oral LD50 of 200 mg/kg BW has also been reported for chlordane toxicity to the rat (WHO 1984). Dogs fed diets containing 0.3, 3, 15, or 30 mg/kg of technical chlordane for 2 years exhibited liver abnormalities in the 15 and 30 mg/kg groups. There were no adverse effects on behavior, appearance, survival, weight gain, or blood chemistry at the lower doses (WHO 1984). In an acute study, a single oral dose of 200 to 700 mg/kg BW/day was reported as a NOAEL (mortality endpoint) for chlordane toxicity to the domestic dog. However, chronic oral doses between 5 and 200 mg/kg BW/day resulted in dose-dependent mortality, with all test animals dying within 25 days to 93 weeks (WHO 1984). In another study, with the exception of the highest concentrations, rats fed diets containing 0.3, 3, 15, 30, or 60 mg/kg of technical chlordane

F-1

for three generations exhibited no measurable effect on fertility, number of young produced, growth, or mortality rate. There were also no gross or microscopic differences among treatment groups up to the 30 mg/kg dose. At 60 mg/kg, the second F_3 generation litters had elevated mortality (11 percent) during the latter part of the nursing period. These animals also exhibited gross and microscopic pathology (WHO 1984).

To express these doses in units of mg/kg BW/day, these values were multiplied by the food ingestion rate for a rat (.015 kg/day) and divided by the body weight of a rat (0.2 kg) to yield the following dietary exposure concentrations:

Dose (mg/kg)	Dose (mg/kg BW/day)
30 (NOAEL, chronic)	1.8
60 (LOAEL, chronic)	3.6

Therefore, a NOAEL of 1.8 mg/kg BW/day and a LOAEL of 3.6 mg/kg BW/day were used in this risk assessment for the evaluation of the dietary toxicity of chlordane to the selected mammalian receptors.

F.1.2   Methoxychlor

F.1.2.1   Chronic Methoxychlor Toxicity to Birds

No literature pertaining to the dietary toxicity of methoxychlor to the red-winged blackbird or the green heron were found. Therefore, literature pertaining to the dietary toxicity of methoxychlor to other bird species was reviewed. Acute oral LC50s of >5,000 mg/kg, dry weight, were reported for the bobwhite, Japanese quail, and pheasant (Heath et al. 1972). Using an accepted correction factor of 10, these doses equate to a LOAEL of 500 mg/kg and a NOAEL of 50 mg/kg, dry weight. Assuming that the diet was 1/3 solids, this equates to a LOAEL of 166.7 mg/kg and a NOAEL of 16.7 mg/kg, wet weight. To express these doses in units of mg/kg BW/day, these values were multiplied by the food ingestion rate of the respective species (bobwhite 0.02 kg/day, Japanese quail 0.01 kg/day, pheasant 0.06 kg/day) and divided by the body weight of the respective species (bobwhite 0.178 kg, Japanese quail 0.09 kg, pheasant 0.953) to yield the following dietary exposure concentrations:

Dose (mg/kg)	Dose (mg/kg BW/day)
16.7 (NOAEL, pheasant)	1.05
16.7 (NOAEL, Jap. quail)	1.86
16.7 (NOAEL, bobwhite)	1.87
166.7 (LOAEL, pheasant)	10.5
166.7 (LOAEL, Jap. quail)	18.6
166.7 (LOAEL, bobwhite)	18.7

A LOAEL of 10.5 mg/kg BW/day, wet weight, and a NOAEL of 1.05 mg/kg BW/day, wet weight, were used in this risk assessment to evaluate the dietary toxicity of methoxychlor to the red-winged blackbird and the green heron.

One study was found in which the dietary toxicity of methoxychlor to mallard ducks was examined. An acute oral LD50 of >2,000 mg/kg was reported for 3-month old mallard ducks (n = 3) (Hudson et al. 1984). Using an accepted conversion factor of 10, this dose equates to a LOAEL of >200 mg/kg and a NOAEL of >20 mg/kg. To express these doses in units of mg/kg BW/day, these values were multiplied by the food ingestion rate of a mallard duck (0.25 kg/day) and divided by the lowest mean reported body weight of a mallard duck

(1.043 kg) to yield dietary exposure concentrations of 48 mg/kg BW/day (LOAEL) and 4.8 mg/kg BW/day (NOAEL).

A LOAEL of 48 mg/kg BW/day and a NOAEL of 4.8 mg/kg BW/day were used in this risk assessment to evaluate the dietary toxicity of methoxychlor to the mallard duck.

F.1.2.2   Chronic Methoxychlor Toxicity to Mammals

No literature pertaining to the toxicity of methoxychlor to the raccoon or red fox could be found. Only one study evaluating the dietary toxicity of methoxychlor to a mammal, the rabbit, was found in the literature. An oral dose of 5.01 mg/kg of methoxychlor for 7 to 19 days during gestation resulted in an excessive loss of litters (U.S. EPA 1991). Using an accepted conversion factor of 10, this dose results in a NOAEL of 0.5 mg/kg. To express these doses in units of mg/kg BW/day, these values were multiplied by the food ingestion rate of a rabbit (0.06 kg/day) and divided by the body weight of a rabbit (2 kg) to yield dietary exposure concentrations of 0.15 mg/kg BW/day (LOAEL) and 0.015 mg/kg BW/day.

A LOAEL of 0.15 mg/kg BW/day and a NOAEL of 0.015 mg/kg BW/day were used in this risk assessment to evaluate the dietary toxicity of methoxychlor to the raccoon and the red fox.

F.1.3   Dieldrin

F.1.3.1   Chronic Dieldrin Toxicity to Birds

Three studies were found in which the toxic effects of dieldrin to mallard ducks was evaluated. In one study, exposure of mallard ducks (*Anas platyrhynchos*) to dietary concentrations of dieldrin ranging from 4 to 30 mg/kg dieldrin (0.36 to 2.7 mg/kg BW/day) for 75 days resulted in a decrease in the biogenic amines serotonin, norepinephrine, and dopamine (Sharma et al. 1976). However, due to the nature of the endpoints evaluated in this study, toxicity studies evaluating endpoints with more ecological significance using other bird species were used in this risk assessment to evaluate the dietary toxicity of dieldrin to the mallard duck.

No studies were found in which the dietary toxicity of dieldrin to the red-winged blackbird or the green heron were evaluated. Therefore, studies in which the dietary toxicity of dieldrin to other bird species were reviewed. Adverse reproductive effects were observed in pheasants exposed to a concentration of 25 and 50 mg/kg dieldrin (4.3 and 8.75 mg/kg BW/day) in their diet (Genelly and Rudd 1956). Hungarian partridges exposed to 3 mg/kg dieldrin (0.5 mg/kg BW/day) in their diet for 90 days during the breeding season resulted in decreased egg production and hatchability (Neill et al. 1969). Chickens exposed to 5 mg/kg dieldrin (0.9 mg/kg BW/day) in their diet showed no effects on egg production or hatchability (Graves et al. 1969). It was estimated that the lowest observed adverse effect level of dieldrin in brown pelican (*Pelecanus occidentalis*) eggs is approximately 1 mg/kg (0.3 mg/kg BW/day) in their diet (Blus 1982). Eggshells of normal thickness were laid by pheasants fed a diet containing approximately 0.1 mg/kg BW/day dieldrin (Dahlgren and Linder 1974).

A LOAEL 0.3 mg/kg BW/day and a NOAEL of 0.1 mg/kg BW/day will be used in this risk assessment to evaluate the dietary toxicity of dieldrin to the red-winged blackbird, the mallard duck, and the green heron.

F.1.3.2   Chronic Dieldrin Toxicity to Mammals

No studies were found in which the dietary toxicity of dieldrin to the raccoon or red fox were evaluated. Therefore, studies in which the dietary toxicity of dieldrin to other mammal species were reviewed.

In one study, rats of varying ages (28 to 750 days old) were exposed to dietary concentrations of dieldrin ranging from 0.08 to 40 mg/kg (0.0003 to 2.4 mg/kg BW/day) (Harr et al. 1970). The exposure resulted in nonspecific neural and vascular lesions, cranial edema, and convulsions at the greater concentrations; no effects were noted at dietary concentrations less than 2.1 mg/kg (0.13 mg/kg BW/day). In a 128-week study, no adverse effects were noted in mice exposed to 0.1 and 1 mg/kg dieldrin (0.013 and 0.13 mg/kg BW/day) in their diet (Walker et al. 1972). In a similar study, no effect on mortality or longevity was observed in three generations of rats exposed to 2.5, 12.5, or 25.0 mg/kg dieldrin in the diet (0.15, 0.75, and 1.5 mg/kg BW/day); however, an increase in the liver/body weight ratio was observed at all concentrations (Treon and Cleveland 1955).

For the purposes of this risk assessment, a NOAEL of 1.5 mg/kg BW/day and a LOAEL of 15 mg/kg BW/day (using an accepted conversion factor of 10) will be used to evaluate the dietary toxicity of dieldrin to the raccoon and the red fox.

F.1.4   Endrin/Endrin Aldehyde/Endrin Ketone

F.1.4.1   Chronic Endrin/Endrin Aldehyde/Endrin Ketone Toxicity to Birds

No literature was found pertaining to the dietary toxicity of endrin to the red-winged blackbird or the green heron. Therefore, literature pertaining to the dietary toxicity of endrin other bird species was reviewed. Acute oral LD50s of 14, 14, and 18 mg/kg, dry weight, were reported for the bobwhite, pheasant, and Japanese quail, respectively (Heath et al. 1972). Using an accepted conversion factor of 10, these doses equate to LOAELs of 1.4, 1.4, and 1.8 mg/kg, dry weight, respectively, and NOAELs of 0.14, 0.14, and 0.18 mg/kg, dry weight, respectively. Assuming that the diet was 1/3 solids, this equates to LOAELs of 0.47, 0.47, and 0.6 mg/kg, wet weight, respectively, and NOAELs of 0.047, 0.047, and 0.06 mg/kg, wet weight, respectively. To express these doses in units of mg/kg BW/day, these values were multiplied by the food ingestion rate of the respective species (bobwhite 0.02 kg/day, pheasant 0.06 kg/day, Japanese quail 0.01 kg/day) and divided by the body weight of the respective species (bobwhite 0.178 kg, pheasant 0.953 kg, Japanese quail 0.09 kg) to yield the following dietary exposure concentrations:

Dose (mg/kg)	Dose (mg/kg BW/day)
0.047 (NOAEL, pheasant)	0.003
0.047 (NOAEL, bobwhite)	0.005
0.06 (NOAEL, Jap. quail)	0.007
0.47 (LOAEL, pheasant)	0.03
0.47 (LOAEL, bobwhite)	0.05
0.6 (LOAEL, Jap. quail)	0.07

A LOAEL of 0.03 mg/kg BW/day, wet weight, and a NOAEL of 0.003 mg/kg BW/day, wet weight, were used in this risk assessment to evaluate the dietary toxicity of endrin and its derivatives to the red-winged blackbird and the green heron.

One study was found which examined the dietary toxicity of endrin to the mallard duck. An acute oral LD50 of 5.64 mg/kg was reported for 12-month old female mallard ducks (Hudson et al. 1984). Using an accepted conversion factor of 10, this dose equates to a LOAEL of 0.564 mg/kg and a NOAEL of 0.0564 mg/kg. To express these doses in units of mg/kg BW/day, these values were multiplied by the food ingestion rate of a mallard duck (0.25 kg/day) and divided by the lowest mean reported body weight of a mallard duck (1.043 kg) to yield dietary exposure concentrations of 0.14 mg/kg BW/day (LOAEL) and 0.014 mg/kg BW/day (NOAEL).

A LOAEL of 0.14 mg/kg BW/day and a NOAEL of 0.014 mg/kg BW/day were used in this risk assessment for the evaluation of the dietary toxicity of endrin and its derivatives to the mallard duck.

F.1.4.2 Chronic Endrin/Endrin Aldehyde/Endrin Ketone Toxicity to Mammals

No literature pertaining to the dietary toxicity of endrin or its derivatives to the raccoon or the red fox was found. Therefore, literature pertaining to the dietary toxicity of endrin and/or its derivatives to other mammals was reviewed. One study was found which examined the dietary toxicity of endrin to the dog. A dietary concentration of 4 mg/kg of endrin for 2 years resulted in few convulsions in the test animals and slight pathology. A dietary concentration of 3 mg/kg resulted only in an increase in heart and liver weight rations (Hayes and Laws, n.d.). In another study, a concentration of 25 mg/kg of endrin in the diet of male and female rats for 2 years resulted in increased mortality in the females. Dietary concentrations of 5 mg/kg for 2 years resulted only in an increase in liver weight ratios in males and kidney weight ratios in females (Hayes and Laws, n.d.). To express these doses in units of mg/kg BW/day, these values were multiplied by the food ingestion rate for a rat (0.015 kg/day) and divided by the body weight of a rat (0.25 kg) to yield dietary exposure concentrations of 1.5 mg/kg BW/day (LOAEL) and 0.3 mg/kg BW/day (NOAEL).

A LOAEL of 1.5 mg/kg BW/day and a NOAEL of 0.3 mg/kg BW/day were used in this risk assessment to evaluate the dietary toxicity of endrin and its derivatives to the raccoon and the red fox.

F.1.5 Heptachlor/Heptachlor epoxide

F.1.5.1 Chronic Heptachlor/Heptachlor epoxide Toxicity to Birds

No literature was found pertaining to the dietary toxicity of heptachlor/heptachlor epoxide to the red-winged blackbird or the green heron. Therefore, literature pertaining to the dietary toxicity of heptachlor/heptachlor epoxide to other bird species was reviewed. Acute oral LC50s of 92, 93, and 224 mg/kg, dry weight, were reported for the bobwhite, Japanese quail, and pheasant, respectively (Heath et al. 1972). Using an accepted conversion factor of 10, these doses equate to LOAELs of 9.2, 9.3, and 22.4 mg/kg, respectively, and NOAELs of 0.92, 0.93, 2.24 mg/kg, respectively. Assuming that the diet was 1/3 solids, this equates to LOAELs of 3.1, 3.1, and 7.5 mg/kg, wet weight, respectively, and NOAELs of 0.31, 0.31, and 0.75 mg/kg, wet weight, respectively. To express these doses in units of mg/kg BW/day, these values were multiplied by the food ingestion rate of the respective species (bobwhite 0.02 kg/day, Japanese quail, 0.01 kg/day, pheasant 0.06 kg/day) and divided by the body weight of the respective species (bobwhite 0.178 kg, Japanese quail 0.09 kg, pheasant 0.953 kg) to yield the following dietary exposure concentrations:

Dose (mg/kg)		Dose (mg/kg BW/day)
0.307 (NOAEL, bobwhite)		0.03
0.31 (NOAEL, Japanese quail)		0.03
0.75 (NOAEL, pheasant)		0.05
3.1 (LOAEL, bobwhite)		0.35
3.1 (LOAEL, Japanese quail)		0.34
7.5 (LOAEL, pheasant)		0.47

A LOAEL of 0.34 mg/kg BW/day, wet weight, and a NOAEL of 0.03 mg/kg BW/day, wet weight, were used in this risk assessment to evaluate the dietary toxicity of heptachlor/heptachlor epoxide to the red-winged blackbird and the green heron.

One study was found which examined the dietary toxicity of heptachlor/heptachlor epoxide to the mallard duck. An acute oral LC50 of 480 mg/kg, dry weight, in feed was reported for the mallard duck (Heath et al. 1972). Using an accepted conversion factor of 10, this dose equates to a LOAEL of 4.8 mg/kg and a NOAEL of 0.48 mg/kg. Assuming that the diet was 1/3 solids, this equates to a wet weight LOAEL of 1.6 mg/kg and a wet weight NOAEL of 0.16 mg/kg. To express these doses in units of mg/kg BW/day, these values were multiplied by the food ingestion rate of a mallard duck (0.25 kg/day) and divided by the lowest mean body weight of a mallard duck (1.043 kg) to yield dietary exposure concentrations of 0.4 mg/kg BW/day (LOAEL) and 0.04 mg/kg BW/day (NOAEL).

A LOAEL of 0.4 mg/kg BW/day, wet weight, and a NOAEL of 0.04 mg/kg BW/day, wet weight, were used in this risk assessment to evaluate the dietary toxicity of heptachlor/heptachlor epoxide to the mallard duck.

F.1.5.2   Chronic Heptachlor/Heptachlor epoxide Toxicity to Mammals

No literature was found pertaining to the dietary toxicity of heptachlor/heptachlor epoxide to the raccoon Therefore, literature pertaining to the dietary toxicity of heptachlor/heptachlor epoxide to another omnivorous mammal, the rat, was reviewed. Only one study was found which examined the dietary toxicity of heptachlor/heptachlor epoxide to the rat. Dietary exposure to heptachlor at 3 mg/kg for 2 years resulted only in an increase in rat liver weights (U.S. EPA 1991). Using an accepted conversion factor of 10, this dose equates to a LOAEL of 30 mg/kg. To express these doses in units of mg/kg BW/day, these values were multiplied by the food ingestion rate for a rat (.015 kg/day) and divided by the body weight of a rat (0.25 kg) to yield dietary exposure concentrations of 18 mg/kg BW/day (LOAEL) and 1.8 mg/kg BW/day (NOAEL).

A LOAEL of 18 mg/kg BW/day and a NOAEL of 1.8 mg/kg BW/day were used in this risk assessment to evaluate the dietary toxicity of heptachlor/heptachlor epoxide to the raccoon.

One study was found which examined the dietary toxicity of heptachlor/heptachlor epoxide to the fox. The minimum amount of dietary heptachlor which caused toxic symptoms and death in wild-collected foxes (unspecified species) over 12 days was 105 mg/kg (Blackmore 1963). Using an accepted conversion factor of 10, this dose equates to a NOAEL of 10.5 mg/kg. To express these doses in units of mg/kg BW/day, these values were multiplied by the food ingestion rate of a fox (0.432 kg/day) and divided by the body weight of a fox (2.7 kg) to yield a LOAEL of 16.8 mg/kg BW/day and a NOAEL of 1.68 mg/kg BW/day .

A LOAEL of 16.8 mg/kg BW/day and a NOAEL of 1.68 mg/kg BW/day were used in this risk assessment to evaluate the dietary toxicity of heptachlor/heptachlor epoxide to the red fox.

F.1.6   DDT/DDD/DDE

F.1.6.1   Chronic DDT/DDD/DDE Toxicity to Birds

No literature was found pertaining to the dietary toxicity of DDT or any of its derivatives to the red-winged blackbird or any other insectivorous bird. Therefore, literature pertaining to the dietary toxicity of DDT to other passerine bird species was reviewed. One study was found which examined the dietary toxicity of DDT and DDE separately to a passerine bird species. Bengalese finches (*Lonchura striata*) were fed diets contaminated with DDT and DDE separately from 6 weeks prior to pairing until fledging of the young had occurred. Ingestion of diets containing 8 mg/kg DDT (equivalent to 2.49 mg/kg BW/day) reduced fertility, hatchability, and fledging success, while ingestion of diets containing 4 mg/kg DDE (equivalent to 1.25 mg/kg BW/day) caused the same effects (Jefferies 1971). Using an accepted conversion factor of 10, the latter dose equates to a NOAEL of 0.125 mg/kg BW/day.

A LOAEL of 1.25 mg/kg BW/day and a NOAEL of 0.125 mg/kg BW/day were used in this risk assessment to evaluate the dietary toxicity of DDT and its derivatives to the red-winged blackbird.

One study was found which examined the acute dietary toxicity of DDT, DDD, and DDE separately to the mallard duck. Acute dietary LD50's of 1,869 mg/kg, 4,814 mg/kg, and 3,572 mg/kg dry weight, were reported for DDT, DDD, and DDE, respectively (Heath et al. 1972). Another study was found in which the toxicity of DDE to a closely related species, the black duck, was evaluated. In this study, black duck hens fed 10 mg/kg, dry weight, (equivalent to 3 mg/kg, wet weight) DDE laid eggs with shells 22 percent thinner at the equator, 30 percent thinner at the cap, and 33 percent thinner at the apex than those of the controls (Longcore and Samson 1973). To express this dose in units of mg/kg BW/day, this value was multiplied by the food ingestion rate of the black duck (0.05 kg/day) and divided by the lowest reported body weight of a black duck (0.9 kg) to yield a LOAEL of 0.17 mg/kg BW/day. Using an accepted conversion factor of 10, this correlates to a NOAEL of 0.017 mg/kg BW/day.

A LOAEL of 0.17 mg/kg BW/day, wet weight, and a NOAEL of 0.017 mg/kg BW/day, wet weight, were used in this risk assessment to evaluate the dietary toxicity of DDT and its derivatives to the mallard duck.

No literature was found pertaining to the dietary toxicity of DDT or any of its derivatives to the green heron. Therefore, literature pertaining to the dietary toxicity of DDT and its derivatives to other carnivorous bird species was reviewed. An acute oral LC50 of >1,200 mg/kg BW was reported for the sandhill crane (Hudson et al. 1984). Wiemeyer and Porter (1970) report mortality in American kestrels receiving 2.8 mg/kg DDE for 14 to 16 months. To express this dose in units of mg/kg BW/day, this value was multiplied by the food ingestion rate of an American kestrel (0.03 kg/day) and divided by the lowest reported mean body weight of an American kestrel (0.103 kg) to yield a dietary exposure concentration of 0.81 mg/kg BW/day. Female American kestrels maintained on a diet containing 0.55 mg/kg BW/day DDE produced eggs with shells that were 15.1 percent thinner than experimental controls (Lincer 1975). Eggshell thinning to the extent reported may be expected to impact

breeding success, since Anderson et al. (1969) states that whenever eggshell thinning exceeds 10 percent, that bird population would be in a decline.

For the purposes of this risk assessment, a LOAEL of 0.55 mg/kg BW/day and a NOAEL of 0.055 were selected to evaluate the toxicity of DDT and its derivatives to the green heron.

F.1.6.2   Chronic DDT/DDD/DDE Toxicity to Mammals

No literature was found pertaining to the dietary toxicity of DDT or its derivatives to the raccoon. Therefore, literature pertaining to the dietary toxicity of DDT, DDD, and DDE to another omnivorous mammal, the rat, was reviewed and used to predict the effects of DDT and its derivatives on the raccoon. Liver damage was observed in rats at a dietary DDT concentration of 500 mg/kg (for 2.9 years) but not at 25 mg/kg (2 years) (Rossi et al. 1977; Treon and Cleveland 1955). No mortality was reported in the 500 mg/kg treatment. In contrast to these numbers, an acute oral LD50 of 118 mg/kg is reported for the rat (Omer 1970), and a single oral dose of 250 mg/kg is reported to be lethal (Galley 1952). DDD was found to be less acutely toxic, with an acute oral LD50 of >4,000 mg/kg for the rat (Gaines 1969). When DDE was administered in the diet at 200 mg/kg for 6 weeks, the rats exhibited differential degrees of humoral and cellular immune suppression (Banerjee et al. 1996). In another study, virgin female Sprague-Dawley rats were given daily doses of 10 mg/kg BW of DDE for 5 days/week for 5 weeks prior to mating. This dose was continued throughout gestation and lactation periods and was not toxic to the dams nor did it have a pronounced effect on neonatal mortality. Furthermore, no significant differences in lactation parameters were observed between DDE-treated groups and the control group (Kornbrust et al. 1986). In another study, however, male and female Osborne-Mendel rats fed DDE at concentrations of 41.95 and 21.85 mg/kg BW/day (males) and 23.1 and 12.1 mg/kg BW/day (females) for 78 weeks exhibited increased mortality in both sexes. In addition, the DDE was hepatotoxic and induced centrolobular necrosis and fatty metamorphosis (NCI/DHEW 1978).

For the purposes of this risk assessment, a LOAEL of 12.1 mg/kg BW/day and a corresponding NOAEL of 1.21 mg/kg BW/day (using an accepted converstion factor of 10) were used to evaluated the effects of DDT and its derivatives to raccoons.

No literature was found pertaining to the dietary toxicity of DDT to the red fox. Therefore, literature pertaining to the dietary toxicity of DDT and its derivatives to another canid, the domestic dog, was reviewed. In one study, no effect on the liver was observed in dogs receiving 400 mg/kg of DDT in their diet for 39 to 49 months, however, minor liver damage was seen at a dietary concentration of 2,000 mg/kg (Lehman 1965). In another study, an acute oral LC50 of 60 mg/kg DDT was reported for the domestic dog (ESE, Inc. 1989). In a study using DDE, a single feeding of 50 mg/kg BW of o,p'-DDE resulted in a complete inhibition of ATPase in the homogenate and the microsomal fraction of the adrenal cortex. Two feedings at this dose caused activation of the enzymes in the homogenate and the mitochondrial fraction (Komissarenko et al. 1997). In a study evaluating the effects of DDD, dogs receiving 50 to 200 mg/kg BW/day of DDD for 1 to 30 months exhibited weakness, anorexia, and mortality (Nelson and Woodard 1949).

For the purposes of this risk assessment, a LOAEL of 50 mg/kg BW/day and a NOAEL of 5 mg/kg BW/day (using an accepted conversion factor of 10) were used to evaluate the dietary toxicity of DDT and its derivatives to the red fox.

F.1.7   PCBs

F.1.7.1   Chronic PCB Toxicity to Birds

One study was found in which the chronic toxicity of PCBs to the mallard duck was evaluated. Aroclor 1254 was fed to 9 month-old mallard hens at a concentration of 25 mg/kg, dry weight, in the diet for at least one month prior to egg laying, and no detrimental effects on reproduction or nest attentiveness were observed (Custer and Heinz 1980). Assuming that the diet was 1/3 solids, this equates to a wet weight concentration of approximately 8.3 mg/kg. To convert this dosage to units of mg/kg BW/day, the dose was first multiplied by the food ingestion rate for the mallard duck (0.25 kg/day), and then divided by the lowest reported adult body weight (1.043 kg) to yield a dose of approximately 2.0 mg/kg BW/day.

Therefore, for the purposes of this risk assessment, a NOAEL of 2.0 mg/kg BW/day and a LOAEL of 20 mg/kg BW/day (using an accepted conversion factor of 10) were used to assess the potential risk posed by PCBs to the mallard duck.

No studies were found in the literature in which the chronic dietary toxicity of PCBs to the green heron was evaluated. Therefore, studies on the toxicity of PCBs to other bird species were reviewed and used to assess the toxicity of PCBs to the green heron.

One study was found in which the toxicity of PCBs to the red-winged blackbird was evaluated. In this study, a dietary concentration of 1500 mg/kg (dry weight) was administered to red-winged blackbirds for 6 days, by which time 50% of the birds had died (Stickel et al. 1984). However, due to the acute nature of this study (short duration and high mortality), this study was not used to assess the chronic effects of PCBs to the red-winged blackbird. Therefore, studies on the toxicity of PCBs to other bird species were reviewed and used to estimate the toxicity of PCBs to the red-winged blackbird.

Delayed reproduction was reported in ringed turtle doves fed a diet of 10 parts per million (ppm) Aroclor 1254 for 3 months (Heinz et al. 1984). Torre and Peterle (1983) investigated the behavioral component of reproduction in mourning doves given dietary supplements of 0, 10, or 40 ppm Aroclor 1254. Control doves displayed normal courtship behaviors and patterns. Doves that were fed at the 10 ppm supplemental level spent twice as much time in the courtship phase as the control birds, with only 50% completing courtship and nesting. Of the 50% that did nest and incubate eggs, nest initiation was significantly delayed. None of the doves on the 40 ppm dietary supplement completed the nesting process. In a different study, hatchability of chicken eggs was reduced in hens fed a diet which was supplemented with 20 ppm of total PCBs; reproductive impairment was observed at supplemental dietary levels as low as 5 ppm (Heinz et al. 1984).

American kestrels fed a diet of 9-10 mg/kg BW/day of Arochlor 1254 for a period of 62-69 days, showed a marked decrease in sperm concentration (Bird et al. 1983). In a separate study, male and female pairs of American kestrels were fed diets containing 3 mg/kg, wet weight, of Aroclor 1248 incorporated into a commercial diet for approximately 20 weeks. Eggs were collected from the pairs 2 to 4 days after egg-laying was complete. The eggs collected from the treated pairs of birds exhibited a significant 5 percent reduction in eggshell thickness (Lowe and Stendell 1991). Neither the body weights nor the food ingestion rates were reported in this study, therefore values from a different study were used to convert the 3 mg/kg dose into a exposure concentration to be used in this risk assessment. The 3 mg/kg dose was divided by the lowest reported mean adult American kestrel body

weight of 0.103 kg and a food ingestion rate of 0.03 kg/day (U.S. EPA 1993) to yield an exposure concentration of 0.87 mg/kg BW/day.

For the purposes of this risk assessment, a LOAEL of 0.87 mg/kg BW/day and a corresponding NOAEL of 0.087 mg/kg BW/day (using an accepted conversion factor of 10) were used to assess the risk of PCBs to the red-winged blackbird and the green heron.

F.1.7.2 Chronic PCB Toxicity to Mammals

No studies were located in which the toxicity of PCBs to the red fox was investigated. One study was found in which the dietary toxicity of PCBs to the raccoon investigated. In this study, an 8-day LD50 of > 50 mg Arochlor 1254/kg in the diet was calculated (Montz et al. 1982). Due to the short-term exposure duration and the endpoint evaluated (50% mortality), this study was not used in this risk assessment to evaluate the chronic effects of PCBs to the raccoon. However, several studies were found pertaining to the dietary toxicity of PCBs to mink, most of which examined its effects on reproduction, growth and survival. Since mink have been identified as one of the most sensitive organisms to the effects of PCBs (Giesy et al. 1994), the dietary toxicity of PCBs to mink will be used to estimate the dietary toxicity of PCBs to the raccoon and the red fox in this risk assessment.

In mink, reproductive effects are seen at parent dietary levels as low as 0.13 mg/kg BW/day (Heaton et al. 1995) and embryotoxicity at parent dietary levels of 0.66 mg/kg BW/day (Aulerich and Ringer 1977). Some adult mortality and behavioral effects are seen at dietary levels starting at 0.148 mg/kg BW/day (Platanow and Karstad 1973), reduced adult weight at dietary levels starting at 1.31 mg/kg BW/day (Aulerich and Ringer 1977), and complete adult mortality at dietary levels starting at 3.3 mg/kg BW/day (Aulerich and Ringer 1977).

Male and female ranch-bred mink were acclimated to a diet consisting of ocean fish scraps, commercial mink cereal, and meat by-products. Ocean fish scraps made up 40 percent of this diet. Dietary treatment levels were prepared by substituting 10, 20, and 40 percent of the ocean fish scraps with PCB-contaminated carp. The mean dietary PCB concentrations were 0.015 mg/kg (control), 0.72 mg/kg (10 percent carp), 1.53 mg/kg (20 percent carp), and 2.56 mg/kg (40 percent carp). Groups of 15 mink (3 males, 12 females) were assigned to one of the four treatment groups for a period of 12 weeks. Mink receiving the highest PCB-containing diet (40 percent carp or 0.32 mg/kg BW/day, as reported by the investigators) exhibited a 42 percent reduction in mean litter size, 86 percent fewer live kits at birth, and no kits surviving beyond 24-hours post-partum. Even mink receiving the 10 percent carp diet (or 0.13 mg/kg BW/day, as reported by the investigators) exhibited a 67 percent reduction in kits surviving three to six weeks relative to the control (Heaton et al. 1995).

One-year-old mink were fed a diet of beef and cereal prepared from cows which had been given 10 consecutive daily oral doses of 1 and 10 mg/kg of Aroclor 1254 dissolved in an olive oil and dairy concentrate (Platanow and Karstad 1973). The cows did not exhibit any clinical, gross, or histopathological signs of PCB toxicity. The cows were killed 24 hours following the last dose, and the musculature, liver, and kidneys ground and mixed with commercial mink food cereal at a level of 24 percent cereal. The resulting rations containing 0.64 and 3.57 mg/kg of total PCB were fed to mink for a period of 160 days. The mink were fed this diet *ad libitum* 2 months prior to the breeding season and continued for 160 days. All 16 mink that were fed 3.57 mg/kg of PCBs died by day 105. Two of the 16 mink that were fed 0.64 mg/kg died by days 122 and 129. The mink exhibited poor appetites, lethargy, and weakness before dying. Some passed tarry feces, indicating gastrointestinal

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hemorrhaging. At both treatment levels, males survived longer than females. These doses were converted to a daily exposure concentration by multiplying them with the inverse of the lowest reported body weight of the mink (0.52 kg) and the food ingestion rate of the mink (0.121 kg/day). This yielded exposure concentrations of 0.148 and 0.785 mg/kg BW/day for the 0.64 and 3.57 mg/kg dose, respectively.

Eight month old mink fed a basal diet containing 1.0 mg/kg of Aroclor 1254 for a period of approximately six months exhibited no mortality or any significant changes in the thyroid, pituitary, adrenal glands, or serum T3 and T4 levels (Wren et al 1987a). Reproduction and kit development was evaluated under the same test conditions in a separate study (Wren et al. 1987b) by the same investigators. Male fertility and female offspring production were not affected by the 1.0 mg/kg Aroclor 1254 diet. However, growth rate of kits nursed by exposed mothers was significantly reduced. The investigators estimated the daily exposure concentrations to be 0.10 mg/kg BW/day for males and 0.18 mg/kg BW/day for females.

In a preliminary study to determine the cause of reproductive complications in mink fed Great Lakes fish, adult breeder mink were fed a basal diet supplemented with 30 mg/kg of PCBs for six months (181 days). However, all of the mink died emaciated by the end of the experimental period (Aulerich and Ringer 1977). For this risk assessment, the 30 mg/kg dose was converted to a daily exposure concentration by multiplying it with the inverse of the lowest reported body weight for the mink (0.52 kg) and the food ingestion rate (0.121 kg/day) to yield an exposure concentration of 6.6 mg/kg BW/day.

As a result of this preliminary study, a long-term study was conducted to ascertain the effects of long-term, low-level consumption of PCBs on growth. Mink were fed a basal diet supplemented with 5 and 10 mg/kg of PCBs for a period of approximately 8.5 months. The basal diet plus 10 mg/kg of PCBs resulted in a significant 56 percent decrease in body weight gain after a period of 4 months. Body weight gain was reduced by 39 percent in the 5 mg/kg treatment group, but this reduction was not significant. Both the 5 and 10 mg/kg treatment groups failed to produce offspring; the control group produced 17 live and 8 dead kits. Various degrees of embryotoxicity were observed during necropsy of the treated animals (Aulerich and Ringer 1977). The 5 and 10 mg/kg doses were converted to a daily exposure concentration by multiplying it with the inverse of the lowest body weight reported by the investigators for this treatment group (0.923 kg) and the food ingestion rate (0.121 kg/day) of the mink. This yielded exposure concentrations of 0.66 and 1.31 mg/kg BW/day for the 5 and 10 mg/kg treatment group, respectively.

Based on the results of this experiment, another experiment was conducted to determine the effects of long-term consumption of low-level PCBs on reproduction. Fifteen mg/kg of PCB as Aroclor 1254 in the diet resulted in a complete inhibition of reproduction and 31 percent adult mortality, compared to 6 percent mortality in the controls. Five mg/kg of Aroclor 1254 resulted in a 95 percent reduction in the number of kits born live; the ratio of live kits to female adults was reduced by 87 percent. However, in an effort to determine the persistency of the impaired reproductive condition, 11 adult females that received 5 mg/kg of Aroclor 1254 for a period of six months were placed on a control diet for one year. The results indicate that the impaired reproductive performance of these females was not a permanent condition (Aulerich and Ringer 1977). The 5 and 15 mg/kg dose was converted to a daily exposure concentration by multiplying it with the inverse of the lowest reported body weight for the mink [and the food ingestion rate (0.121 kg/day)] to yield exposure concentrations of 1.1 and 3.3 mg/kg BW/day, respectively.

For the purposes of this risk assessment, a LOAEL of 0.13 mg/kg BW/day and a NOAEL of 0.10 mg/kg BW/day were used to estimate the toxicity of PCBs to the raccoon and the red fox.

F.1.8 Arsenic

F.1.8.1 Chronic Arsenic Toxicity to Birds

One study was found in which the toxicity of arsenic to the mallard duck was evaluated. In this study, no effects on reproduction were observed in mallard ducks exposed to three dose levels of sodium arsenite up to 10 mg/kg BW/day for a period of 112 days (Stanley et al. 1994).

Therefore, a NOAEL of 10 mg/kg BW/day and a LOAEL of 100 mg/kg BW/day (using an accepted conversion factor of 10) were used in this risk assessment to assess the toxicity of arsenic to the mallard duck.

No studies pertaining to the dietary toxicity of arsenic to the red-winged blackbird or green heron were found. Therefore, literature studies pertaining to the dietary toxicity of arsenic to other avian species, including the study summarized above, were reviewed. A single oral dose of an organoarsenical compound was used to develop an LD50 of 47.6 mg/kg BW/day for the California quail (Hudson et al. 1984) and 33 mg/kg BW/day for the chicken (NAS 1979).

Because of the reproductive endpoint and the longer duration of exposure, the mallard duck study was used to establish NOAEL and LOAEL values for the red-winged blackbird and green heron. A NOAEL of 10 mg/kg BW/day and an estimated LOAEL of 100 mg/kg BW/day will be used to evaluate the risk to these receptors.

F.1.8.2 Chronic Arsenic Toxicity to Mammals

No studies pertaining to the dietary toxicity of arsenic to the raccoon or red fox were found. Therefore, literature pertaining to the dietary toxicity of arsenic to other mammals was reviewed.

The National Resources Council of Canada (NRCC 1978) states that mammals in general have oral LD50 values ranging from 10 to 50 mg/kg of lead arsenate. A study conducted on mice indicated an oral LD50 of 39.4 mg/kg BW/day after 96 hours (NAS 1979). Adverse reproductive effects were noted in a study where mice were administered a chronic oral dose as low as 1.26 mg/kg BW/day (Schroeder and Mitchner 1971). Toxicity was noted in a study using cats administered an oral dose of 1.5 mg/kg BW/day (Pershagen and Vahter 1979).

Based on these results, a LOAEL of 1.26 mg/kg BW/day and an estimated NOAEL of 0.126 mg/kg BW/day arsenic will be used to evaluate the risk posed by arsenic to the selected mammalian receptors.

F.1.9 Cadmium

F.1.9.1 Chronic Cadmium Toxicity to Birds

A variety of studies were found in which the dietary toxicity of cadmium to the mallard duck

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was examined. In one study, juvenile mallard drakes were fed diets containing 0, 50, 150, or 450 mg/kg of Cd for 42±1 days (Di Giulio and Scanlon 1984). The most significant metabolic effects were seen only in the 450 mg/kg (103.5 mg/kg BW/day) treatment group. These specimens exhibited a 20.3 percent decrease in body weight, a 26 percent decrease in liver weight, a 15 percent increase in kidney weight, a 21 percent decrease in liver aldolase activity, a 46 percent increase in plasma uric acid concentrations, a 74 percent decrease in plasma triiodothyronine (T_3) concentrations, a 28 percent increase in adrenal weights, and a 31 percent increase in adrenal cortisone concentrations. Ducks in the 150 mg/kg (34.5 mg/kg BW/day) treatment group also exhibited a 12 percent increase in kidney weight and a 23 percent increase in adrenal weight. No adverse effects were observed at a dietary concentration of 50 mg/kg (11.5 mg/kg BW/day).

In another study, mallard ducklings were fed 0, 5, 10, or 20 mg/kg of Cd in diet from day 1 of age to 12 weeks of age (Cain et al. 1983). Ducklings receiving 20 mg/kg (4.6 mg/kg BW/day) exhibited an 8 percent decrease in packed cell volume, a 6 percent reduction in hemoglobin concentration, and a 52 percent increase in serum glutamic pyruvic transaminase activity, all of which were statistically significant at the 0.05 level. Necropsies of these specimens revealed mild to severe kidney lesions. Whole body Cd concentrations were 58.30 to 65.30 mg/kg, dry weight. No significant adverse effects were noted at a dietary Cd level of 5 mg/kg (1.2 mg/kg BW/day) or 10 mg/kg (2.3 mg/kg BW/day).

Finally, adult (1 year old) male and female mallard ducks were fed a diet containing 0.08, 1.6, 15.2, and 210 mg/kg, wet weight, of Cd as cadmium chloride *ad libitum* for 90 days (White and Finley 1978). The corresponding daily dietary intake as provided by the investigators was 0.018 mg/kg BW/day (0.08 mg/kg), 0.37 mg/kg BW/day (1.6 mg/kg), 3.5 mg/kg BW/day (15.2 mg/kg), and 48.3 mg/kg BW/day (210 mg/kg). Male testis weight, male kidney weights, and egg production by females were significantly less in the 210 mg/kg (48.3 mg/kg BW/day) treatment when compared to the controls. No adverse effects were observed at a dietary concentration of 15.2 mg/kg (3.5 mg/kg BW/day).

Due to the ecological significance of the egg production endpoint of the latter study, a LOAEL of 48.3 mg/kg BW/day and a NOAEL of 15.2 mg/kg BW/day were used in this risk assessment to assess the effects of cadmium on the mallard duck.

No studies pertaining to the dietary toxicity of cadmium to the red-winged blackbird or green heron were found. Therefore, literature pertaining to the dietary toxicity of cadmium to other bird species will be used as surrogates to assess the dietary toxicity of cadmium to the red-winged blackbird and green heron. In addition to the studies using mallard ducks, summarized above, a variety of other birds species have been used to examine the toxicity of cadmium. In one study, male Japanese quail fed a diet containing 75 mg/kg (12 mg/kg BW/day) of Cd as $CdCl_2$ for 4 weeks exhibited a 62 percent decrease in testis size, a lack of spermatogenesis, damage to small intestine mucosa, and severe anemia (Richardson et al. 1974). These responses were associated with a mean liver Cd concentration of 42±2.6 mg/kg. A 5-day exposure/3-day post-exposure LC_{50} of 1,496 mg Cd/kg was calculated for this species by another investigator (Hill and Camardese 1986).

White Plymouth rock hens fed a diet containing 12 mg/kg (2.1 mg/kg BW/day) of Cd as $CdSO_4$ $8H_2O$ for 48 weeks exhibited a significant ($p<0.05$) 25 percent decrease in egg production associated with a mean muscle Cd concentration of 6.46 mg/kg, dry weight (Leach et al. 1979) Similarly, domestic hens fed a diet containing 50 mg Cd/kg (8.75 mg/kg BW/day) and 100 mg Cd/kg (17.5 mg/kg BW/day) also exhibited reduced egg production and egg weight (Anke et al. 1970). A concentration of 200 mg Cd/kg (4.4 mg/kg BW/day)

resulted in ceased egg production within 2 days.

Juvenile (2-week old) male Leghorn chickens were fed a diet *ad libitum* containing various concentrations of Cd in two separate experiments (Pritzl et al. 1974). Experiment 1 utilized Cd concentrations of 0, 400, 600, 800, and 1,000 mg/kg for 20 days. Experiment 2 utilized Cd concentrations of 0 and 700 mg/kg for 20 days. A significant reduction in growth rate and feed consumption was noted at a dietary concentration of 400 mg/kg (40 mg/kg BW/day). An LD_{50} of 565 mg/kg (56.5 mg/kg BW/day) was calculated from Experiment 2. In a separate study, Leghorn chickens were fed a diet containing cadmium sulfate for a period of 48 weeks. Eggshell thickness and egg production were unaffected at levels as high as 0.97 mg/kg BW/day (Leach et al. 1979).

This latter study was used to develop the NOAEL and LOAEL values for the red-winged blackbird and the green heron because of the long exposure period and the ecological significance of the endpoints. A NOAEL of 0.97 mg/kg BW/day and a LOAEL of 9.7 mg/kg BW/day (using an accepted converstion factor of 10) for cadmium were used to evaluate the effects of the red-winged blackbird and the green heron in this risk assessment.

F.1.9.2   Chronic Cadmium Toxicity to Mammals

No studies pertaining to the dietary toxicity of cadmium to the raccoon or red fox were found. Therefore literature pertaining to the dietary toxicity of cadmium to the rat was reviewed. The rat will be used as a surrogate to assess the dietary toxicity of cadmium to the raccoon and the red fox.

Male and female weanling brown rats (O.S.U. strain) fed a diet containing 5 mg/kg (0.75 mg/kg BW/day) of Cd as $CdCl_2$ for 10 weeks exhibited no adverse effects on growth (Pribble and Weswig 1973). No effects on reproduction of adult Norway rats exposed to four dose levels of cadmium chloride for a period of six weeks were noted at concentrations as high as 1 mg/kg BW/day (Sutou et al. 1980).

For this risk assessment, the latter study was used to derive the NOAEL and LOAEL because of the range of concentrations evaluated and the ecological significance of the endpoints. A dietary exposure level of 1 mg/kg BW/day was used as a NOAEL for the selected mammalian receptors. A LOAEL of 10 mg/kg BW/day was derived from this NOAEL using an accepted conversion factor of 10.

F.1.10   Lead

F.1.10.1   Chronic Lead Toxicity to Birds

No studies pertaining to the dietary toxicity of lead to the mallard duck or the green heron were found in the literature. One study on the effects of lead to the red-winged blackbird was found, in which a lethal dosage of lead acetate was administered in the diet to the birds. It was found that blood protoporphyrin decreased, ALAD increased, and renal intranuclear inclusion bodies were present prior to death (Beyer, et al. 1988). However, due to the high lethal dosage and the experimental design (4.2 mg/kg BW/day, increased by 60% each week until 50% of the birds were dead), this study will not be used to derive a LOAEL to estimate the toxicity of lead to the red-winged blackbird. Therefore, literature pertaining to the dietary toxicity of lead to other bird species was reviewed and used to assess the chronic dietary toxicity of lead to the red-winged blackbird, mallard duck, and green heron.

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The gastric motility of adult male and female red-tailed hawks fed 0.82 and 1.64 mg Pb/kg BW/day in a single oral dose was evaluated through the use of surgically implanted transducers for a period of 3 weeks following the dose. Neither concentration had any effect on gastric contractions or egestion of undigested material pellets (Lawler et al. 1991).

A study conducted on red-tailed hawk found that 3 mg/kg/day of lead caused the clinical symptoms of lead poisoning (Reiser and Temple 1981). A similar study found that 3 mg/kg/day fed to starlings caused a reduction in muscle condition and altered their feeding activity (Osborn et al. 1983). Adult male and female red-tailed hawks given an oral dose of 0.82 mg/kg BW/day each day for 3 weeks resulted in an 83 percent decrease in delta-aminolevulinic acid dehydratase activity and a 74 percent increase in the levels of free porphyrins circulating in the blood (Redig et al. 1991). Edens et al. (1976) exposed Japanese quail to 4 oral dose levels of lead acetate for a period of 12 weeks. The study identified a NOAEL of 0.133 mg/kg BW/day for egg production and a LOAEL of 1.33 mg/kg BW/day for hatching success.

The results of the latter study will be used to develop the NOAEL and LOAEL values based on the ecological significance of the endpoints and the method and duration of exposure. A LOAEL of 1.33 mg/kg BW/day and a NOAEL of 0.133 mg/kg BW/day were used to evaluate the risk posed by lead to the selected avian receptors.

F.1.10.2   Chronic Lead Toxicity to Mammals

No controlled dietary toxicity studies on lead using the raccoon or red fox were found in the literature. Therefore, literature pertaining to the dietary toxicity of lead to other mammals was reviewed and will be used to assess the chronic dietary toxicity of lead to the raccoon and red fox.

Mason and MacDonald (1986) evaluated the effect of lead and cadmium on otter (*Lutra lutra*). Daily lead intake was estimated on the basis of measured fecal lead levels, the known ingestion rate for otter, and gastrointestinal lead absorption rates for mammals. Estimated lead intake correlated well with levels measured in major fish prey species. No apparent impact on population levels was found when lead intake was less than 0.15 mg/kg BW/day whereas otter populations were reduced in areas where the estimated lead intake exceeded 2 mg/kg BW/day. Adult pregnant mice (C57Bl strain) were fed a diet containing lead concentrations of 0.125, 0.25, 0.5, and 1 percent for 48 hours after mating (Jacquet et al. 1976). Dietary lead concentrations of 0.125 percent (16 mg/kg BW/day), 0.25 percent (32 mg/kg BW/day), and 0.5 percent (64 mg/kg BW/day) resulted in an increase in the number of embryos in the 4-cell stage versus the 8-cell stage. A delayed effect of increased non-divided embryos resulted from a dietary lead concentration of 1 percent (128 mg/kg BW/day). Azar et al. (1973) administered lead to rats in 5 dietary levels for three generations and measured changes in reproduction and growth. A dosage of 80 mg/kg BW/day reduced offspring weights and produced kidney damage in the young, while a dosage of 8 mg/kg BW/day did not result in adverse effects.

For this risk assessment, the latter study was used to select NOAEL and LOAEL values because of the ecological significance of the endpoints, the range of dose levels selected, and the duration of the study. A dietary exposure level of 8 mg/kg BW/day was used as a NOAEL and 80 mg/kg BW/day was used as a LOAEL to evaluate the risk posed by lead to the selected mammalian receptors.

F.1.11 Mercury

F.1.11.1 Chronic Mercury Toxicity to Birds

No studies pertaining to the dietary toxicity of mercury to the red-winged blackbird, mallard duck, or green heron were found. Therefore, literature pertaining to the dietary toxicity of mercury to other bird species was reviewed. These other bird species will be used as surrogates to assess the dietary toxicity of mercury to the red-winged blackbird, mallard duck, and green heron.

Starlings fed 0.1 mg/kg BW/day of Hg for 8 weeks were observed to have kidney lesions (Nicholson and Osborn 1984). Zebra finches fed a diet containing 1.75 mg Hg/kg BW/day suffered from neurological impairment and death while finches exposed to 0.88 mg Hg/kg BW/day had no signs of mercury poisoning (Scheuhammer 1988). Red-tailed hawks fed a diet containing 1.12 mg Hg/kg BW/day suffered from mortality, dilatation of myelin sheaths and loss of myelin. Hill and Schaffner (1976) exposed Japanese quail to five dose levels of mercuric chloride for a period of one year and identified a NOAEL of 0.60 mg/kg BW/day for egg production, fertility and hatching. Goshawks exposed to doses ranging from 0.7 to 1.2 mg/kg BW/day suffered complete mortality after between 30 and 47 days of exposure (Borg et al. 1970).

For this risk assessment, the latter study was used to estimate risk of mercury to the selected avian receptors. A dietary level of 0.7 mg/kg BW/day was used as a LOAEL. A NOAEL of 0.07 mg/kg BW/day was derived from this LOAEL using an accepted conversion factor of 10.

F.1.11.2 Chronic Mercury Toxicity to Mammals

No studies pertaining to the dietary toxicity of mercury to the raccoon or red fox were found in the literature. Therefore, studies pertaining to the dietary toxicity of mercury to other mammals were reviewed.

Rats that were fed a diet containing 0.5 mg Hg/kg BW/day showed reduced fertility (Khera 1979). In a similar study rats fed a diet containing 0.32 mg/kg BW/day showed no adverse effects (ATSDR 1993). Mink that were fed a diet containing 0.58 mg CH_3Hg/kg BW/day for a period of 100 days showed no effects (Jernelov et al. 1976). Aulerich (1974) exposed ranch mink to one oral dose level of mercuric chloride for a period of five months. The study identified a NOAEL of 1.0 mg/kg BW/day for reproduction and mortality. In another study, adult female mink were fed diets containing 1.1, 1.8, 4.8, 8.3 and 15 mg/kg Hg as methyl mercuric chloride for 93 days, and signs of intoxication (anorexia, weight loss, ataxia, splaying of hind legs, irregular vocalization, and convulsions) were evident in the 1.8 mg/kg and higher treatment groups, while no clinical signs were observed in the 1.1 mg/kg treatment group (Wobeser et al. 1975). Using an ingestion rate of 0.16 g/g BW/day and a body weight of 974 g for adult female mink (U.S. EPA 1993), this equates to a LOAEL of 0.29 mg/kg BW/day and a NOAEL of 0.18 mg/kg BW/day. Dogs exposed to 0.1 mg Hg/kg BW/day during pregnancy showed a higher than normal incidence of stillbirths (Khera 1979).

For the purposes of this risk assessment, the latter study was used to assess the hazards of mercury to the selected mammalian receptors. A LOAEL of 0.1 mg/kg BW/day Hg and a NOAEL of 0.01 mg/kg BW/day Hg (using an accepted conversion factor of 10) were used to evaluate the effects of mercury to the raccoon and red fox.

F.1.12 Selenium

F.1.12.1 Chronic Selenium Toxicity to Birds

Only one study was found in which the dietary toxicity of selenium was evaluated in birds. The bird species used in this study was the mallard duck. Therefore, this study was used to estimate the toxic effects of selenium to all three avian receptors, the red-winged blackbird, the mallard duck, and the green heron. In this study, mallard ducks were given diets containing 100 mg/kg of inorganic selenite usually died within a month. Birds receiving 25 mg/kg continued to survive for over 3 months, but growth and reproduction were dramatically reduced. Teratogenic effects were evident at 3 months in mallard ducklings receiving 10 mg/kg. Ducks and their young appeared normal after 3 months exposure to 5 mg/kg of dietary selenite (Eisler 1985).

To express the 5 and 10 mg/kg exposure concentration in units of mg/kg BW/day, this value was multiplied by the food ingestion rate for a mallard duck (0.25 kg/day) and divided by the lowest reported mean body weight for a mallard duck (1.043 kg) to yield body weight-normalized exposure concentration of 1.2 and 2.4 mg/kg BW/day.

For this risk assessment, a LOAEL of 2.4 mg/kg BW/day and a NOAEL of 1.2 mg/kg BW/day were used to estimate the risk of selenium to the red-winged blackbird, mallard duck, and the green heron.

F.1.12.2 Chronic Selenium Toxicity to Mammals

No studies pertaining to the dietary toxicity of selenium to the raccoon or the red fox were found in the literature. Therefore, literature pertaining to the dietary toxicity of selenium to the rat was reviewed. The rat will be used as a surrogate to assess the dietary toxicity of selenium to the raccoon and the red fox.

Rats fed diets containing 0.8 to 1.0 mg/kg during lifetime exposure developed intestinal lesions (Eisler 1985). To express 0.8 mg/kg in units of mg/kg BW/day, this value was multiplied by the food ingestion rate of a rat (0.015 kg/day) and the inverse body weight of a rat (1/0.25 kg) to yield a body weight-normalized exposure concentration of 0.048 mg/kg BW/day.

For the purposes of this risk assessment, a LOAEL of 0.048 mg/kg BW/day and a NOAEL of 0.0048 mg/kg BW/day (using an accepted conversion factor of 10) were used to assess the toxicity of selenium to the raccoon and the red fox.

F.2 Acute Toxicity Profiles for the Food Chain Model

F.2.1 PCBs

F.2.1.1 Acute PCB Toxicity to Birds

The green heron was the only measurement endpoint for which a chronic hazard quotient of greater than one was obtained when PCBs were evaluated in the food chain model using the chronic LOAEL reference value derived above. Therefore, the green heron was the only avian measurement endpoint which was evaluated for the potential of PCBs to pose an acute risk. No literature information was found pertaining to the acute dietary toxicity of PCBs to

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the green heron. Therefore, literature pertaining to the acute dietary toxicity of PCBs to other bird species was reviewed.

In a five-day acute dietary toxicity study, LD50's (expressed as mg/kg in the diet) for Arochlor 1248 were calculated to be 1,175 mg/kg for the Northern bobwhite, 2,798 mg/kg for the Mallard duck, 1,312 mg/kg for the ring-necked pheasant, and 4,844 mg/kg for the Japanese quail. For Arochlor 1254, LD50's were calculated to be 604 mg/kg for the Northern bobwhite, 2,699 mg/kg for the Mallard duck, 1,091 mg/kg for the ring-necked pheasant, and 2,898 mg/kg for the Japanese quail (Heath et al. 1972). To express these dosages in units of mg/kg body weight/day, these values were multiplied by the food ingestion rate of the respective specie (bobwhite 0.02 kg/day, Mallard duck 0.25 kg/day, Japanese quail, 0.01 kg/day, pheasant 0.06 kg/day) and divided by the body weight of the respective species (bobwhite 0.178 kg, Mallard duck 1.043 kg, Japanese quail 0.09 kg, pheasant 0.953 kg) to yield the following dietary exposure concentrations:

Dose (mg/kg)	Dose (mg/kg BW/day)
1,175 (Arochlor 1248 LD50, bobwhite)	132
2,798 (Arochlor 1248 LD50, Mallard)	671
1,312 (Arochlor 1248 LD50, pheasant)	82.6
4,844 (Arochlor 1248 LD50, quail)	538
604 (Arochlor 1254 LD50, bobwhite)	67.8
2,699 (Arochlor 1254 LD50, Mallard)	647
1,091 (Arochlor 1254 LD50, pheasant)	68.6
2,898 (Arochlor 1254 LD50, quail)	322

Therefore, for the purposes of this risk assessment, an acute LD50 of 67.8 mg/kg BW/day was used to assess the acute toxicity of PCBs to the green heron.

F.2.1.2   Acute PCB Toxicity to Mammals

A chronic hazard quotient of greater than one was obtained when PCBs were evaluated in both the raccoon and red fox food chain models using the chronic LOAEL reference values derived above. Therefore, both the raccoon and the red fox were evaluated for the potential of PCBs to pose an acute risk. One study was found in which the dietary toxicity of PCBs to the raccoon investigated. In this study, an 8-day LD50 of > 50 mg Arochlor 1254/kg in the diet was calculated (Montz et al. 1982). However, since lethality was not observed in this study, or any of the other studies which were found in which the acute dietary toxicity of PCBs was evaluated, studies which determined acute oral LD50's of Arochlor mixtures administered orally to rats were used to evaluate the acute dietary toxicity of PCBs in both the raccoon and the red fox.

In single dosage experiments using rats, the acute oral LD50 of Arochlor 1248 was found to be between 800 and 11,000 mg/kg BW, and the acute oral LD50 of Arochlor 1254 was found to be between 500 and 1,400 mg/kg BW (Eisler 1986). Therefore, for the purposes of this risk assessment, an acute oral LD50 of 500 mg/kg body weight was used to evaluate the acute toxicity of PCBs to the raccoon and the red fox.

F.2.2   Lead

F.2.2.1   Acute Lead Toxicity to Birds

The mallard duck was the only measurement endpoint for which a chronic hazard quotient of

greater than one was obtained when lead was evaluated in the food chain model using the chronic LOAEL reference value derived above. Therefore, the mallard duck was the only avian measurement endpoint which was evaluated for the potential of lead to pose an acute risk.

A variety of toxicity studies have been performed in which lead, in the form of lead shot, was administered to mallard ducks and acute lethality was observed. In one such study, a single oral dose of 1.4 g of lead shot was administered to mallard ducks and some deaths were observed (Longcore et al. 1974a). When 1.0 g of lead shot was administered to mallards, 9% mortality was observed within 20 days (Longcore et al. 1974b). To convert these two dosages to units of mg/kg BW, they were divided by the lowest reported body weight for an adult mallard duck of 1.043 kg (EPA 1993) to yield dosages of 1,342 mg/kg BW and 959 mg/kg BW, respectively. Dieter and Finley (1978) reported that when a single oral dose of 205 mg of lead shot (reported by the authors to be equivalent to 151 mg/kg BW), some deaths were observed.

Since lead shot is a specific form of lead which is unlikely to be present within the Bound Brook stream corridor, additional literature was also reviewed in which other forms of inorganic lead were examined for their acute toxicity to other avian species. In one study, when American kestrel nestlings were dosed orally with metallic lead powder daily for 10 days, a dosage of 625 mg/kg BW was reported to cause 40% mortality in six days (Hoffman et al. 1985). In another study, a single oral dose of 75 mg Pb/kg BW, as lead acetate, was reported to cause mortality (Kendall and Scanlon 1985).

Therefore, for the purposes of this risk assessment, an acute oral dosage of 75 mg Pb/kg BW was used to evaluate the acute toxicity of lead to the mallard duck.

F.2.3   Selenium

F.2.3.1   Acute Selenium Toxicity to Mammals

The raccoon was the only measurement endpoint for which a chronic hazard quotient of greater than one was obtained when selenium was evaluated in the food chain model using the chronic LOAEL reference value derived above. Therefore, the raccoon was the only mammalian measurement endpoint which was evaluated for the potential of selenium to pose an acute risk. No literature information was found pertaining to the acute dietary toxicity of selenium to the raccoon. Therefore, literature pertaining to the acute dietary toxicity of selenium to other mammalian species was reviewed.

An oral LD50 of 6700 mg/kg BW has been reported for rats (RTECS 1997). In sheep fed plant material containing selenium, death was noted at concentrations of 3.2 to 12.8 mg Se/kg body weight (Eisler 1985). The minimum lethal oral dose of selenite was found to be 3.3 mg Se/kg body weight for horses and mules to 11 mg Se/kg BW for cattle and 15 mg Se/kg BW for swine (Eisler 1985).

Therefore, for the purposes of this risk assessment, an acute dietary dose of 3.3 mg/kg body weight was used to evaluate the acute toxicity of selenium to the raccoon.

F.3   Toxicity of Whole Body Tissue Residue Levels

F.3.1   a-Chlordane/g-Chlordane

F.3.1.1   a-Chlordane/g-Chlordane Toxicity to Crayfish

No literature was found pertaining to toxic chlordane residue levels in freshwater aquatic macroinvertebrates. However, estuarine invertebrates exhibited reduced survival at tissue concentrations of 106 mg/kg, wet weight, lipid-normalized (Zitko 1978). Since the analytical data from the crayfish collected for this risk assessment are not lipid-normalized, the residue level of 106 mg/kg, was multiplied by the average percent lipid concentration (2.4%) measured in the crayfish collected for this risk assessment, and divided by 100 to yield a toxicity threshold for chlordane of 2.5 mg/kg, wet weight, in whole body crayfish tissue.

F.3.1.2   a-Chlordane/g-Chlordane Toxicity to Fish

Chlordane residue levels of 300 to 4,000 mg/kg, wet weight, lipid-normalized, were associated with mortality in fish (Zitko 1978). To be conservative, the lower level of 300 mg/kg was used to compare with tissue levels of chlordane in fish collected from the site. However, since the analytical data from the fish collected for this risk assessment are not lipid-normalized, the residue level of 300 mg/kg was multiplied by the average percent lipid concentration (3%) measured in the forage fish (whole bodies) collected for this risk assessment, and divided by 100 to yield a toxicity threshold for chlordane of 9 mg/kg, wet weight, in whole body fish tissue.

F.3.1.3   a-Chlordane/g-Chlordane Toxicity to Small Mammals

No studies were found linking whole body chlordane levels to toxicity in small mammals.

F.3.2   Methoxychlor

F.3.2.1   Methoxychlor Toxicity to Crayfish

No studies were found linking whole body methoxychlor leves to toxicity in aquatic macroinvertebrates.

F.3.2.2   Methoxychlor Toxicity to Fish

No studies were found linking whole body methoxychlor levels to toxicity in fish.

F.3.2.3   Methoxychlor Toxicity to Small Mammals

No studies were found linking whole body methoxychlor levels to toxicity in small mammals.

F.3.3   Dieldrin

F.3.3.1   Dieldrin Toxicity to Crayfish

No studies were found linking whole body dieldrin levels to toxicity in aquatic macroinvertebrates.

F.3.3.2 Dieldrin Toxicity to Fish

No studies were found linking whole body dieldrin levels to toxicity in fish.

F.3.3.3 Dieldrin Toxicity to Small Mammals

No studies were found linking whole body dieldrin levels to toxicity in small mammals.

F.3.4 Endrin/Endrin Aldehyde/Endrin Ketone

F.3.4.1 Endrin/Endrin Aldehyde/Endrin Ketone Toxicity to Crayfish

No studies were found linking whole body levels of endrin or any of its derivatives to toxicity in aquatic macroinvertebrates.

F.3.4.2 Endrin/Endrin Aldehyde/Endrin Ketone Toxicity to Fish

No studies were found linking fish fillet or whole body levels of endrin or any of its derivatives to toxicity in fish.

F.3.4.3 Endrin/Endrin Aldehyde/Endrin Ketone Toxicity to Small Mammals

No studies were found linking whole body levels of endrin or any of its derivatives to toxicity in small mammals.

F.3.5 Heptachlor/Heptachlor Epoxide

F.3.5.1 Heptachlor/Heptachlor Epoxide Toxicity to Crayfish

No studies were found linking whole body levels of heptachlor or heptachlor epoxide to toxicity in aquatic macroinvertebrates.

F.3.5.2 Heptachlor/Heptachlor Epoxide Toxicity to Fish

No studies were found linking fish fillet or whole body levels of heptachlor or heptachlor epoxide to toxicity in fish.

F.3.5.3 Heptachlor/Heptachlor Epoxide Toxicity to Small Mammals

No studies were found linking whole body levels of heptachlor or heptachlor epoxide to toxicity in small mammals.

F.3.6 DDT/DDD/DDE

F.3.6.1 DDT/DDD/DDE Toxicity to Crayfish

No studies were found linking whole body levels of DDT or any of its derivatives to toxicity in aquatic macroinvertebrates.

F.3.6.2 DDT/DDD/DDE Toxicity to Fish

No studies were found linking fish fillet or whole body levels of DDT or any of its derivatives to toxicity in fish.

F.3.6.3 DDT/DDD/DDE Toxicity to Small Mammals

No studies were found linking whole body levels of DDT or any of its derivatives to toxicity in small mammals.

F.3.7 PCBs

F.3.7.1 PCB Toxicity to Crayfish

Although no studies were located in which whole body PCB concentrations were linked to toxicity in crayfish, a variety of studies have been performed in which whole body concentrations of PCBs were linked to toxicity in other macroinvertebrates. These studies are summarized in Beyer et al. (1996). In summary the lowest whole body concentration considered by Beyer et al. (1996) to adversely affect macroinvertebrate survival, growth, or reproduction is 25 mg/kg, wet weight. For the purposes of this risk assessment, a whole body concentration of 25 mg/kg, ww, will be used as a threshold concentration to assess the effects of PCBs on crayfish.

F.3.7.2 PCB Toxicity to Fish

A variety of studies have been performed in which whole body concentrations of PCBs in fish were linked to toxicity in fish. These studies are summarized in Beyer et al. (1996). In summary the lowest whole body concentration considered by Beyer et al. (1996) to adversely affect fish survival, growth, or reproduction is 50 mg/kg, wet weight. This concentration will be used in this risk assessment to assess the effects of PCBs on fish.

F.3.7.3 PCB Toxicity to Small Mammals

No studies were found linking whole body PCB levels to toxicity in small mammals.

F.3.8 Arsenic

F.3.8.1 Arsenic Toxicity to Crayfish

No studies were found linking whole body arsenic levels to toxicity in aquatic macroinvertebrates.

F.3.8.2 Arsenic Toxicity to Fish

No studies were found linking whole body arseniclevels to toxicity in fish.

F.3.8.3 Arsenic Toxicity to Small Mammals

No studies were found linking whole body arsenic levels to toxicity in small mammals.

F.3.9 Cadmium

F.3.9.1 Cadmium Toxicity to Crayfish

No studies were found linking whole body cadmium levels to toxicity in aquatic macroinvertebrates.

F.3.9.2 Cadmium Toxicity to Fish

No studies were found linking whole body cadmium levels to toxicity in fish.

F.3.9.3 Cadmium Toxicity to Small Mammals

No studies were found linking whole body cadmium levels to toxicity in small mammals.

F.3.10 Lead

F.3.10.1 Lead Toxicity to Crayfish

No studies were found linking whole body lead levels to toxicity in aquatic macroinvertebrates.

F.3.10.2 Lead Toxicity to Fish

No studies were found linking whole body lead levels to toxicity in fish.

F.3.10.3 Lead Toxicity to Small Mammals

No studies were found linking whole body lead levels to toxicity in small mammals.

F.3.11 Mercury

F.3.11.1 Mercury Toxicity to Crayfish

No studies were found linking whole body mercury levels to toxicity in aquatic macroinvertebrates.

F.3.11.2 Mercury Toxicity to Fish

A variety of studies were identified in which whole body concentrations of mercury were linked to toxicity in fish. These studies are summarized by Beyer et al. (1996). In summary, whole body concentrations of about 5 mg/kg, wet weight, or greater are associated with toxic effects in brook trout, and in rainbow trout, whole body concentrations of about 10 mg/kg, wet weight, and above seem to be associated with toxic effects. Therefore, a whole body concentration of 5 mg/kg, wet weight, in fish will be used as the threshold concentration to assess the effects of mercury on fish for this risk assessment.

F.3.11.3 Mercury Toxicity to Small Mammals

No literature was found linking whole body mercury levels to toxicity in small mammals.

F.3.12 Selenium

F.3.12.1 Selenium Toxicity to Crayfish

No studies were found linking whole body selenium levels to toxicity in aquatic macroinvertebrates.

F.3.12.2 Selenium Toxicity to Fish

A variety of studies were identified in which whole body concentrations of selenium were linked to toxicity in fish. These studies are summarized by Beyer et al. (1996). In summary, the lowest whole body concentration that was linked to either mortality or reproductive failure in fish was a concentration of 4 mg/kg, dry weight. To convert this to a wet weight concentration, the residue level of 4 mg/kg was multiplied by the average percent solids concentration (21 %) measured in the fish fillets collected for this risk assessment, and divided by 100. The percent solids for fish fillets were used to make this conversion because percent solids data for whole body fish was not available. The resulting concentration, 0.84 mg/kg, wet weight, will be used as a threshold whole body concentration to assess the effects of selenium on fish for this risk assessment.

F.3.12.3 Selenium Toxicity to Small Mammals

No studies were found linking whole body selenium levels to toxicity in small mammals.

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APPENDIX G

Life Histories
Cornell-Dubilier Site
South Plainfield, NJ
April 1998

LIFE HISTORIES

G.1   *Hyalella azteca* as Representative of Benthic Invertebrates

Justification of *Hyalella azteca* as Representative of Benthic Invertebrates

Hyalella azteca were selected as representative benthic invertebrates because they are in direct contact with sediment for a significant portion of their life cycle, they are widely distributed in aquatic systems, they are an important food item for aquatic invertebrate consumers, and they are easy to use in laboratory toxicity evaluations.

Life History (*Hyalella azteca*)

The amphipod, *Hyalella azteca*, is commonly found in freshwater lakes, streams, ponds, and rivers throughout North and South America. In preferred habitats, they are known to reach densities in excess of 10,000 per square meter. They may also be found in sloughs, marshes, and ditches, but generally in lower numbers (U.S. EPA 1994).

Hyalella are epibenthic detritivores that feed on coarse particulate organic material. They typically burrow into surface sediment, and avoid bright light. Because of their feeding and behavioral characteristics, they are ideal test organisms for toxicological evaluation of freshwater sediments. Avoidance of light by movement into the sediment keeps these organisms almost constantly in contact with sediment contaminants (U.S. EPA 1994).

Reproduction in this crustacean is sexual. Males are larger than females and have larger front gnathopods that are presumably used for holding the female during amplexus and copulation. During amplexus, the male and female feed together for a period of up to one week. The pair separates temporarily while the female goes through a molting period. Immediately after the molt, the two rejoin and copulation begins. During copulation, the male releases sperm near the female's marsupium. The female sweeps the sperm into her marsupium, and simultaneously releases eggs from her oviducts into the marsupium where fertilization takes place. The average brood size for female *Hyalella* is 18 eggs per brood, but this number can vary with environmental conditions and physiological stress (U.S. EPA 1994).

Developing embryos and hatched young are kept inside the female's marsupium until she undergoes a second molt. At that time, the juvenile *Hyalella* are released into the surrounding environment. Under favorable conditions, each female produces approximately one brood during every ten day time period (U.S. EPA 1994).

Hyalella have a minimum of 9 instars, with 5 to 8 pre-reproductive stages. The first five stages are juvenile stages; instars 6 and 7 form the adolescent stages; and stages 8 and higher are considered adult (fully reproductive) stages (U.S. EPA 1994).

Exposure Profile for *Hyalella azteca*

Since direct contact with and ingestion of contaminated sediment are the primary routes of exposure for *H. azteca* in the toxicity test, the results of this test were used to indicate both routes of exposure in the risk assessment.

G.2   Red-winged Blackbird as Representative of Insectivorous Birds

<u>Justification for the Selection of the Red-winged Blackbird as Representative of Insectivorous Birds</u>

The red-winged blackbird was selected as representative of insectivorous birds because of its dietary composition, abundance in North America, and preference for moist areas.

<u>Life History</u>

The habitat of the red-winged blackbird is marshes, swamps, wet and dry meadows, and sometimes pastures. Although it is primarily a marsh bird, it will nest near virtually any body of water and occasionally breeds in upland pastures. It breeds from Alaska to Newfoundland south to Florida, the Gulf Coast, and central Mexico. It winters north to Pennsylvania and British Columbia and can often be found in large flocks in the winter (Bull and Farrand 1977; National Geographic Society 1987).

Males generally migrate north prior to females to compete for breeding territory. Those with the best territories attract harems of up to 15 females (Gill 1990). Nests are well-made cups built from marsh grasses or reeds attached to growing marsh vegetation or built in a bush in a marsh. Each pair raises two or three broods a season, building a new nest for each clutch. Each clutch consists of an average of 3 to 5 young (Bull and Farrand 1977). One study found that a Red-winged chick spent 12 days in the egg and 10 more as a nestling (Daniel 1957). Young males do not attain adult male plumage until one year (Gill 1990).

<u>Exposure Profile</u>

An average adult male blackbird weighs 63.6 g, while an average adult female weighs 41.5 g (Clench and Leberman 1978). Based on year-round observations, the diet is composed of 73 percent vegetable matter and 27 percent animal matter (Bent 1965). However, during the breeding season the adult diet is primarily aquatic insects (Orians 1980), and food delivered to nestlings is 97.8 percent animal matter (Snelling 1968).

No ingestion rates were located for red-winged blackbirds; however, an ingestion rate of 8.4 g per day was cited for European blackbirds (Kenaga 1973). For the purposes of this risk assessment, this ingestion rate will be used to represent the ingestion rate of the red-winged blackbird.

Reported territory sizes range from 0.17 acres (Case and Hewitt 1963) to 0.74 acres (Nero 1956). Therefore, it was assumed that a red-winged blackbird could obtain 100 percent of its diet from the contaminated area (area use factor of 1), since the area comprising the on-site sampling locations was approximately 20 acres.

Based on feeding habits, it was assumed that incidental soil/sediment ingestion by red-winged blackbirds would be negligible. A daily water ingestion rate of 7 mL per day was calculated using the following allometric equation derived by Calder and Braun (1983) for water intake by avian species:

$$\text{Water Intake (L/day)} = 0.059 \times \text{Weight (kg)}^{0.67}$$

For this risk assessment, it was assumed that 100 percent of their diet was comprised of insects. However, insects were not available in sufficient quantities for collection and analysis. Crayfish, however, were available in greater quantities than originally anticipated. Therefore, crayfish were used to represent insects for the purposes of this risk assessment.

G.3 Mallard duck (*Anas platyrhynchos*) as Representative of Omnivorous Birds

Justification for the Selection of the Mallard Duck as Representative of Omnivorous Birds

The mallard duck was selected as representative of omnivorous birds because of its dietary composition, abundance in North America, and preference for moist areas.

Life History

The mallard duck is a common surface-feeding duck that is widespread throughout most of the United States. They often interbreed with domestic ducks as well as with black ducks (*Anas rubripes*). The males are generally heavier than the females.

Wintering mallards tend to prefer natural wetlands and rivers over reservoirs and farm ponds. The primary habitat requirement for nesting appears to be dense grassy vegetation at least a half a meter high. Nests are usually located within a few miles of water (U.S. EPA 1993).

In winter, mallards feed primarily on seeds but also on invertebrates, mast, agricultural grains, and, to a lesser extent on leaves, buds, stems, rootlets, and tubers. In spring, the females shift from a largely herbivorous diet to primarily invertebrates in order to obtain enough protein for their molt and subsequent egg production. The animal diet continues throughout the summer as females produce new clutches to replace those that have been destroyed. Ducklings also consume aquatic invertebrates almost exclusively (U.S. EPA 1993).

The mallard winters in all four of the waterfowl flyways of North America (Pacific, Central, Mississippi, and Atlantic); the Mississippi flyway contains the highest numbers. They arrive at their wintering grounds in the Mississippi Valley in mid-September through early November and depart for their northern breeding grounds in March. Adult females that breed successfully are likely to return to the same nesting ground the following year. Each pair of mallards uses a home range. The drake usually establishes a territory which he defends (U.S. EPA 1993).

Nest failure is an important factor affecting mallard populations. Mammalian predation is the main cause of failure, followed by human disturbance. Juvenile survival depends mainly on food and habitat availability. Adult females suffer great natural mortality rates than do males. By fall, there is usually a higher proportion of males than females in most populations. Adult mortality rates are also higher in areas with greater hunting pressure.

Exposure Profile

The lowest reported mean body weight for the mallard duck was 1,225 g for males and 1,043 g for females (U.S. EPA 1993). A food ingestion rate of 0.25 kg/day with a dietary composition of 90 percent plant matter and 5 percent fish was reported for this species (Newell et al. 1987).

A water ingestion rate of 0.055 to 0.058 g/g BW/day (U.S. EPA 1993) was multiplied by the lowest reported mean body weight (1,043 g) to yield a water ingestion rate of 57 to 60 g/day. A sediment ingestion rate of 3.3 percent of the total diet (Beyer et al. 1994) was multiplied by the food ingestion rate (0.25 kg/day) to yield a sediment ingestion rate of 0.008 kg/day.

The home range of this species vary from 111 hectares (274 acres) for a laying female to 620 hectares (1,532 acres) for an adult male (U.S. EPA 1993).

For the purposes of this risk assessment, it was assumed that mallard ducks consume 100% crayfish, since crayfish were collected from the site and analyzed for the COPCs.

G.4   Green Heron as Representative of Piscivorous Birds

Justification for the Selection of the Green Heron as Representative of Piscivorous Birds

The green heron was selected as representative of piscivorous birds because of its dietary composition, abundance in North America, and preference for moist areas.

Life History

The green heron is generally a solitary bird and prefers streams, ponds, and marshes with woodland trees, where it can often be found perching (National Geographic Society 1987). Its range includes British Columbia, Minnesota, and New Brunswick south to southern South America. It winters north to South Carolina, the Gulf Coast, and California (Bull and Farrand 1977).

The green heron builds its nests in thick bushes or trees near water and searches for prey in the soft muddy borders of waterbodies. The structures of their nests are flimsy saucers of sticks and twigs, and 3 to 6 eggs are produced per clutch. The green heron occasionally breeds in colonies (Bull and Farrand 1977).

Exposure Profile

The average body weight of a green heron was reported to be 0.25 kg (Newell et al. 1987). A dietary ingestion rate of 0.047 kg/day was estimated using a regression equation for wading birds (log y = 0.966 log x - 0.640), where x is weight in grams and y is ingestion rate (Kushlan 1978). The diet of the green heron reportedly consists of 44% fish, 21% insects, 24% spiders and miscellaneous invertebrates, and 1% crustaceans (Palmer 1962).

A territorial size of < 2 ha (< 4.9 acres) was reported by Palmer (1962) for the green heron. This was estimated using an average breeding area. The 2 ha area was an average of four group breeding areas ranging in size from 1 to 3.3 ha and containing 8 to 70 breeding pairs. The size of the individual territories were within the group breeding area and varied with habitat and stage of breeding cycle. Therefore, it was assumed that a green heron could obtain 100 percent of its diet from the contaminated area (area use factor of 1), since the area comprising the on-site sampling locations was approximately 20 acres.

Calder and Braun (1983) estimated a water ingestion rate for the green heron of 0.023 L/day based on the allometric equation (water ingestion rate = $0.059 \, Wt^{0.67}$, where Wt is average body weight of the species in kilograms).

An incidental sediment ingestion rate could not be found in the literature. Therefore, to evaluate this exposure pathway, a model was developed that predicted the amount of sediment which may be entrained in the digestive system of bluegill (*Lepomis machrochirus*) and crayfish (*Orconectes* sp.). Fish and crayfish were assumed to be the only food source for the green heron to complete this derivation.

Bluegills commonly reach a size of 12 ounces (Pflieger 1975). From this, the amount of sediment entrained in fish 12 ounces (340 g) in weight was predicted. A study evaluating the stomach contents of 153 bluegills reported an average content of detritus and sediment to be 9.6 percent of the total diet (Kolehmainen 1974). A daily food ingestion rate of 1.75 percent of the body weight per day has been reported for the bluegill (Kolehmainen 1974). This provides a predicted intake rate of 5.95 g of food per day for a 340 g fish. If a conservative assumption is made that 9.6 percent of the food ingested is entirely sediment, it can be predicted that a fish of this size may contain 0.5712 g of sediment in its digestive system.

For the purpose of this model, it was assumed that the level of sediment contained in the digestive system of a fish remains constant over time. This value (0.5712 g) was divided by the predicted fish body weight (340 g) to express sediment entrained in fish digestive systems in units of grams of sediment per gram of fish body weight. This provided a value of 0.00168 g sediment/g body weight. When this value is multiplied by the food ingestion rate of the green heron (48 g/day), the predicted sediment ingestion rate for the green heron through the consumption of fish is approximately 0.08 g/day.

As with the bluegill, life history information for the crayfish (*Orconectes* sp.) was used in predicting the incidental sediment ingestion rate for the green heron via consumption of freshwater invertebrates. Adult *O. virilis* weigh from 5 to over 20 g and consume 0.3 to 1 percent of its total body weight per day (Kim 1994; Tack 1941; Vannote 1963). To express the food ingestion rate in units of g/day, the highest reported food ingestion rate of 1 percent of the total body weight per day was multiplied by the lowest reported body weight of 5 g to yield a food ingestion rate of 0.05 g/day. *Orconectes* spp. detritus ingestion rates range from 10 percent of the total diet per day in young-of-the-year *Orconectes immunis* (Vannote 1963) to 11 percent of the total diet per day in *O. virilis* (Tack 1941). For this risk assessment, it will be assumed that these values represent the percentage of sediment in the diet of a crayfish. The food ingestion rate of 0.05 g/day was multiplied by the incidental sediment ingestion rate of 11 percent of the total diet per day to yield an incidental sediment ingestion rate of 0.0055 g/day.

For the purpose of this model, it was assumed that the level of sediment contained in the digestive system of crayfish remains constant over time. Therefore, to express the amount of sediment entrained in a crayfish's digestive system in units of gram of sediment per gram of crayfish body weight, the sediment ingestion rate of 0.0055 g/day was divided by the lowest adult crayfish body weight of 5 g to yield a sediment ingestion rate of 0.0011 g sediment/g BW of crayfish/day. When this value is multiplied by the food ingestion rate of the green heron (48 g/day), the predicted incidental sediment ingestion rate for the green heron via consumption of crayfish is 0.05 g/day.

For the purposes of the calculation of the incidental sediment ingestion rate for the green heron, the diet will be assumed to consist of 44% fish and 46% crayfish, based on the diet described by Palmer (1962) and assuming that crayfish represent insects, spiders, crustaceans, and miscellaneous invertebrates. Given this dietary ratio, the green heron is estimated to ingest a total of 0.058 g/day of sediment.

For the purposes of the food chain model in this risk assessment, it was assumed that the green heron consumes 100% fish, since fish were collected from the site and analyzed for the COPCs.

G.5   Raccoon (*Procyon lotor*) as Representative of Omnivorous Mammals

Justification for using raccoon as an omnivorous mammal

The raccoon was chosen as an omnivorous mammal based on its opportunistic feeding strategies, its abundance in North America, and the likelihood of its occurrence at the site.

Life History

Raccoons are common and mostly nocturnal mammals inhabiting wooded areas near water, marshes, suburban areas, or virtually any area that can provide food, a den, and permanent water (Hoffmeister 1989; Jones and Birney 1988). Their dens are usually within 1,200 feet from a water supply but are situated in an area where the den can remain dry (Hoffmeister 1989). These dens may be in hollow trees, burrows, caves, crevices in rock, haystacks, chimneys, or under logs (Hoffmeister 1989; Schwartz and Schwartz 1981). During periods of heavy snow or ice, raccoons will den together for several days (Schwartz and Schwartz 1981), otherwise, they are normally solitary and remain active throughout the year (Jones and Birney 1988).

Raccoons are opportunistic omnivores consuming various food items such as berries, fruit, nuts, corn, seeds, aquatic and terrestrial invertebrates, eggs, frogs, snakes, fish, muskrats, and young waterfowl (Jones and Birney 1988; Schwartz and Schwartz 1981). Seasonal and local food availability appears to dictate dietary composition, although as a general rule, plant matter comprises a greater portion of the diet than does animal matter (Barbour and Davis 1974).

Breeding may occur from December through July, although most breeding occurs from January to March (Schwartz and Schwartz 1981; Jones and Birney 1988). Gestation lasts for approximately 63 days with litter sizes ranging from two to seven young (Barbour and Davis 1974). The young are weaned at 10 to 12 weeks, forage with the female parent well into the autumn, and are ready to breed their first winter (Barbour and Davis 1974). Natural predators of the raccoon include owls, hawks, bobcats, coyotes (Merritt 1987; Schwartz and Schwartz 1981). Most raccoons live less than 5 years in the wild (Schwartz and Schwartz 1981).

Exposure Profile

Adult raccoons weigh from 3 to 15 kg (Merritt 1987; U.S. EPA 1993). The home range of this species varies from 12 to 12,350 acres (Merritt 1987).

The food ingestion rate for a raccoon is reported to be approximately 500 g/day (Newell 1987); the water ingestion rate is estimated to be approximately 0.083 g/g BW/day. To express the water ingestion rate in units of g/day, the water ingestion rate of 0.083 g/g BW/day was multiplied by the lowest reported body weight of 3 kg to yield a water ingestion rate of 24.9 g/day (24.9 ml/day).

A soil ingestion rate of 9.4 percent of the total diet has been reported for the raccoon (Beyer *et al.* 1994). To express this value in units of g/day, the soil ingestion rate of 9.4 percent was multiplied by the food ingestion rate of 500 g/day to yield a soil ingestion rate of 47 g/day.

For the purposes of this risk assessment, it was assumed that the raccoon consumes 50% crayfish and 50% fish, since these two types of organisms were collected from the site and analyzed for the COPCs. One exception is for location 6, where crayfish were not available for collection. For this site, it was assumed that 100% of the raccoon's diet is fish.

G.6   Red Fox (*Vulpes vulpes*) as Representative of Carnivorous Mammals

Justification for using red fox as a carnivorous mammal

The red fox was chosen as a carnivorous mammal based on its ingestion of small mammals and its abundance in North America.

Life History

Red fox inhabit open meadows, ditch banks, field and wood edges, fencerows, stream and lake borders, and farmlands (Hoffmeister 1989; Jones and Birney 1988; Merritt 1987). With the exception of the breeding season, red fox have no permanent home but sleep on the ground (Schwartz and Schwartz 1981). A den, usually modified from an existing woodchuck or fox den, is dug during the breeding season and exceptionally cold winters (Barbour and Davis 1974). These scent-marked dens have multiple rooms, entrances, and trails leading to and from hunting areas (Schwartz and Schwartz 1981). In addition to their dens, both males and females will defend their scent-marked hunting territory from intruders (Jones and Birney 1988).

The red fox is primarily an opportunistic carnivore, consuming food items such as rabbits, opossums, muskrats, skunks, rodents, birds, eggs, carrion, invertebrates, snakes, and frogs (Barbour and Davis 1974; Merritt 1987). Some vegetable matter such as fruits and nuts are also consumed when in season (Jones and Birney 1988). During times of abundant food supply, the red fox will bury surplus food to return to for consumption at a later time (Schwartz and Schwartz 1981).

Male and female foxes pair for life, remaining together from midwinter to summer. Females bear one litter per year usually between March and April (Merritt 1987). Gestation periods last from about 49 to 56 days, with most averaging 53 days (Schwartz and Schwartz 1981). The pups are weaned at about 60 days, leave the den in the autumn, and are sexually mature by their first winter (Merritt 1987). Natural predators of the red fox are few but include large hawks and owls, and possibly coyotes (Merritt 1987; Schwartz and Schwartz 1981). Red fox may live from six to ten years in the wild (Schwartz and Schwartz 1981).

Exposure Profile

Adult red fox weigh from 2.7 to 7 kg (Barbour and Davis 1974; Jones and Birney 1988). Home ranges vary from 245 to 1,235 acres (Merritt 1987).

The food ingestion rates of the red fox range from 0.069 g/g BW/day for a nonbreeding adult, to 0.16 g/g BW/day for a juvenile (U.S. EPA 1993). The water ingestion rate for an adult red fox is estimated to be approximately 0.086 g/g BW/day (U.S. EPA 1993). To express these values in units of g/day, the highest reported food ingestion rate of 0.16 g/g BW/day and the water ingestion rate of 0.086 g/g BW/day were multiplied by the lowest reported body weight of 2.7 kg (2,700 g) to yield a food ingestion rate of 432 g/day and a water ingestion rate of 232.2 g/day (232.2 ml/day).

A soil ingestion rate of 2.8 percent of the total diet has been reported (Beyer *et al.* 1994) for the red fox. To express this value in units of g/day, the soil ingestion rate of 2.8 percent was multiplied by the food ingestion rate of 432 g/day to yield a soil ingestion rate of 12.1 g/day.

For the purposes of this risk assessment, it was assumed that the red fox consumes 100% small mammals (specifically white-footed mice), since white-footed mice were collected and analyzed for this risk assessment.

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APPENDIX H

Final Histopathological Evaluation Report
Cornell-Dubilier Site
South Plainfield, NJ
April 1998

NAME/SPECIES: WESTON REAC, UNKNOWN
REQUESTING DR: 03347-142-001-2274-0
ANIMAL ID: ATTN: JOHN JOHNSTON
UNK

WESTON/REAC PROJECT
2890 WOODBRIDGE AVE #209
EDISON, NJ 08837-3679

VETERINARY PATHOLOGY

CASE#: VR-97-001031

INFORMATION

Animal Reference Pathology
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33. Liver & Kidney HISTOPATH

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RESEARCH

VR-97-1031

WESTON REAC STUDY #03347-142-001-2274-01

SLIDE A- (B10401)
KIDNEY- The tissue is acutely congested and well preserved. Significant
changes are not identified.

LIVER- This tissue is acutely congested with good preservation of the
hepatocytes. No significant pathologic lesions are identified.

SLIDE B- (B10402)
KIDNEY- This tissue is acutely congested with normal architecture. The
autolytic change is minimal.
LIVER- This tissue demonstrates multifocal collections of lymphocytes and
plasma cells in the parenchyma with scattered aggregates of parasite eggs.
These eggs have the appearance of Trematode eggs, and they are massive in
numbers. There is some secondary inflammation in focal areas in the portal
triad areas. The number of parasite eggs appears to be extreme throughout all
sections of liver tissue.

SLIDE C- (B10403)
KIDNEY- This tissue is acutely congested with mild autolysis and no
significant inflammation or degeneration.

LIVER- The liver tissue demonstrates multiple granulomata with parasite
eggs. The parasite eggs appear to be Trematode eggs. There are areas of
abscessation with necrosis and degeneration of the hepatic tissue. Trematode
parasites are identified in one section of liver tissue. The fibrosis and
pyogranulomatous inflammation correlates with invasion of the parasites
through the liver, liver damage, and secondary bacterial infection. The
number of parasite eggs and parasites are moderate to severe in this liver.

NAME/SPECIES: WESTON REAC, UNKNOWN
REQUESTING DR: 03347-142-001-2274-0
ANIMAL ID: ATTN: JOHN JOHNSTON
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WESTON/REAC PROJECT
2890 WOODBRIDGE AVE #209
EDISON, NJ 08837-3679

13862643
ARP ID#: (06727)000-22-74
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│      VETERINARY PATHOLOGY        │
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CASE#: VR-97-001031

RESEARCH

SLIDE D- (B10404)
KIDNEY- This tissue is acutely congested and slightly autolyzed. There are
multifocal collections of lymphocytes and plasma cells in the interstitial
areas. Focal areas of hemorrhage support acute trauma or inflammation.
LIVER- The liver section includes small fragments of liver tissue with
scattered parasites or parasite eggs. Minimal other hepatic change is present
in the liver tissue. Mild congestion has occurred in the liver tissue
present. Some of these parasites appear to be nematode parasites. Specific
other change is not identified.

SLIDE E- (B10405)
KIDNEY- This renal tissue is acutely congested with mild autolysis. Minimal
inflammatory cell accumulation has occurred in the interstitium.

LIVER- Multiple sections of liver are submitted with areas of fibrosis and
degeneration. The fibrotic change is irregular. Eosinophils and epithelioid
cells are part of the collection. Epithelioid cells, multinucleated giant
cells and other inflammatory cells are part of the chronic inflammatory
process in the portal triad areas. Fibrosis is limited to the portal triad
areas with hemosiderin and eosinophilia. Some foreign material which is
likely parasitic ova can be found in small granulomas. Eosinophilic
microabscesses support parasitic invasion. Foci of hepatocytic necrosis
suggests chronic passive congestion and secondary degeneration. Specific
other inflammatory or degenerative change is not identified.

SLIDE F- (B10406)
KIDNEY- This section of kidney is acutely congested with minimal autolysis.
Specific degeneration or change is not identified.

LIVER- The liver tissue demonstrates areas of nodular hyperplasia with
multifocal collections of lymphocytes, neutrophils, and plasma cells. There
are a few lymphocytes and neutrophils in portal areas with very thin layers of
fibrous connective tissue stroma. Foci of cellular calcification and
degeneration are present near the capsule, and suggest past parasitic
migration sites.

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VETERINARY PATHOLOGY

CASE#: VR-97-001031

RESEARCH

SLIDE G- (B10407)
KIDNEY- This tissue is acutely congested with mild autolytic change. No
significant lesions are identified.

LIVER- This tissue is acutely congested. There are very few collections of
lymphocytes and plasma cells in the portal triad areas, but no other specific
hepatocellular degeneration or inflammation.

SLIDE H- (B10408)
KIDNEY- This tissue is acutely congested with minimal autolysis. No specific
inflammation is otherwise identified.

LIVER- The liver tissue demonstrates pyogranulomatous inflammation
surrounding many ova. Multinucleated giant cells are part of the
collection. The parasite eggs appear to be Trematode eggs. There are focal
sites suggesting nematodes with chronic inflammation. Pyogranulomatous
inflammation, fibrosis, and degeneration are secondary. Primary liver disease
not associated with parasite eggs cannot be identified.

SLIDE I- (B10409)
KIDNEY- The renal tissue is acutely congested and slightly autolyzed. No
specific inflammation is identified in the renal tissue.

LIVER- The liver demonstrates multifocal areas of marked fibrosis with
chronic lymphocytic plasmacytic and eosinophilic infiltration including
multiple granulomata surrounding many parasite eggs. The parasite eggs are
similar to other parasites identified in this collection of animals. Some
parasites support Trematode organisms as well as the eggs. In one or two
sites, there are suggestions of nematode parasites as well.

SLIDE J- (B10410)
KIDNEY- The renal tissue is acutely congested with no evidence of autolysis.
No specific inflammation or degeneration is identified.

LIVER- This tissue is acutely congested with focal areas of hemorrhage. The
hemorrhage supports a degenerative process, and a traumatic process in the
hepatic tissue. Other significant change or inflammation is not identified.

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RESEARCH

Specific other primary liver disease is not identified.

SLIDE K- (B10411)
KIDNEY- This tissue is acutely congested with mild to moderate autolysis.
Specific other change is not present in the renal tissue.

LIVER- This tissue is slightly autolyzed and very mildly congested. Specific
inflammation or parasites are not identified. The degenerative process
appears to be due to autolysis and not inflammation, infection or infestation
of parasites.

SLIDE L- (B10412)
KIDNEY- This tissue is moderately to severely autolyzed with acute
congestion. Specific inflammation or change is not identified.

LIVER- This tissue is moderately autolyzed and acutely congested. A very few
inflammatory cells are present in the portal triad areas, but they are
definitely minimal in numbers. Evidence of toxicity or parasitic change
cannot be identified. Specific other change is not identified.

SLIDE M- (B10413)
KIDNEY- This renal tissue is acutely congested with minimal autolysis.
Inflammation or degeneration is not readily identified.

LIVER- This tissue is acutely congested. Specific inflammation or other
change cannot be identified in this tissue.

SLIDE N- (B10414)
KIDNEY- The renal tissue is acutely congested. This tissue is slightly
autolyzed with no evidence of specific inflammation or degeneration.

LIVER- The liver tissue is very mildly autolyzed over the surface with acute
congestion of much of the tissue. Specific other change is not identified.

SLIDE O- (B10415)

WESTON REAC, UNKNOWN

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RESEARCH

KIDNEY- The renal tissue is acutely congested with very mild autolysis. Almost no inflammation is identified.

LIVER- This tissue is acutely congested with very minimal autolysis. No evidence of specific degeneration or inflammation is identified. The cellularity is minimally identified.

SLIDE P- (B10416)
KIDNEY- This tissue is acutely congested with mild autolysis. The autolysis is not severe enough to interfere with interpretation. Specific other change is not identified.

LIVER- The liver tissue is acutely congested with focal autolysis where bile has touched the liver surface. A localized aggregate of parasites and parasite eggs is identified in one end of the liver tissue. The number of parasites is mild to moderate. The inflammatory infiltrate in the portal triad areas is very minimal.

SLIDE Q- (B10417)
KIDNEY- This tissue is acutely congested with no significant histologic change.

LIVER- This tissue is acutely congested with autolysis over the surface, but no evidence of any specific inflammation or change in the tissue. Parasites are not present.

SLIDE R- (B10418)
KIDNEY- The renal tissue is acutely congested with very mild autolytic change. No evidence of inflammation or other specific inflammation could be identified.

LIVER- The liver tissue is acutely congested. There is mild autolysis, but no other specific reaction or change is identified.

SLIDE S- (B10419)
KIDNEY- The renal tissue is acutely congested with mild autolysis, but no other specific inflammation or change.

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VETERINARY PATHOLOGY

CASE#: VR-97-001031

RESEARCH

LIVER- This tissue is acutely congested with no significant inflammation or hepatocellular damage.

SLIDE T- (B10420)
KIDNEY- This tissue is acutely congested with mild autolysis. No other significant change is present.

LIVER- This tissue is acutely congested with mild autolysis. No specific inflammatory change or degenerative process is occurring in this liver tissue. There is some evidence of autolysis over the surface, particularly where the gall bladder is contacting the liver tissue.

SLIDE U- (B10421)
KIDNEY- This tissue is acutely congested with no significant histologic change in the renal parenchyma.

LIVER- This tissue is acutely congested with multifocal mild to moderate autolysis. There are foci of neutrophils and fibrinous exudation with necrosis in some parts of the liver parenchyma. Fibrinous exudation has occurred in several foci. The necrosis supports a bacterial infection at this site.

SLIDE V- (B10422)
KIDNEY- This renal tissue is acutely congested. No evidence of degeneration or inflammation is identified.

LIVER- The liver tissue is acutely congested. There is no evidence of specific degeneration or inflammation in any part of this liver tissue.

SLIDE W- (B10423)
KIDNEY- This tissue is acutely congested with no evidence of specific inflammation. Minimal autolysis is identified.

LIVER- The liver tissue is acutely congested. There are multifocal areas of parasite egg accumulation with epithelioid cells and granulation tissue. Mild lymphocytic plasmacytic inflammation has occurred in the portal triad areas.

NAME/SPECIES: WESTON REAC, UNKNOWN
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CASE#: VR-97-001031

RESEARCH

Fibrosis is part of the reactive process.

SLIDE X- (B10424)
KIDNEY- This tissue is acutely congested with mild autolysis and small foci
of lymphocytes and plasma cells collecting in the interstitium. Specific
other change is not present.

LIVER- This tissue is acutely congested with multifocal areas of inflammation
in the portal triads. These areas of inflammation include lymphocytes and
plasma cells. Specific other inflammation or change is not identified.

SLIDE Y- (B10425)
KIDNEY- This tissue is acutely congested with areas of autolysis in the
pelvic tissue. Focal areas of mild lymphocytic plasmacytic inflammation have
occurred in the renal parenchyma.

LIVER- This tissue is acutely congested. Specific inflammation is not
identified. Specific degeneration is not part of the liver tissue.

SLIDE Z- (B10426)
KIDNEY- The renal tissue is acutely congested and slightly autolyzed.
Specific inflammation is not identified.

LIVER- The liver tissue is acutely congested and very mildly autolyzed.
Other specific inflammation is not present.

SLIDE AA- (B10427)
KIDNEY- This tissue is acutely congested with minimal autolysis. No other
specific inflammation is present.

LIVER- This tissue demonstrates multifocal collections of parasite eggs with
fibrosis and mild chronic inflammation around the parasite eggs. The
parasites are the same type of trematodes as previously described in other
animals. The inflammatory process is chronic.

SLIDE AB- (B10428)

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VETERINARY PATHOLOGY

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RESEARCH

KIDNEY- This tissue is very mildly and acutely congested with no evidence of specific degeneration.

LIVER- The liver tissue is acutely and very mildly congested. Mild vacuolization of hepatocytes has occurred in the liver tissue. There are areas of autolysis over the capsule of the liver parenchyma. Other specific change is not identified.

SLIDE AC- (B10429)
KIDNEY- The renal tissue is acutely congested and demonstrates no specific autolysis. Minimal inflammation is identified.

LIVER- The liver tissue is acutely congested with no specific degenerative change of the hepatocytes. Mild autolysis has occurred over the liver capsule.

SLIDE AD- (B10430)
KIDNEY- The renal tissue is acutely congested and mildly autolyzed. Specific inflammation or degeneration is not identified.

LIVER- The liver tissue is acutely congested with multifocal areas of fibrosis with granulation tissue and collections of lymphocytes and plasma cells. Fibrotic change is irregular. The fibrotic change is limited to one area of the liver parenchyma. This site may be an area of past parasite migration. Parasite eggs are not identified at this site, but there is evidence of chronic inflammation.

SLIDE AE- (B10431)
KIDNEY- The renal tissue is acutely congested with no evidence of specific inflammation or alteration.

LIVER- The liver tissue is acutely congested with no evidence of specific change. There is some vacuolization of the hepatocytes, but this is secondary and nonspecific.

SLIDE AF- (B10432)

WESTON REAC, UNKNOWN

NE/SPECIES: WESTON REAC, UNKNOWN
REQUESTING DR: 03347-142-001-2274-0
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CASE#: VR-97-001031

RESEARCH

KIDNEY- This tissue is acutely congested. Mild autolysis has occurred in the renal parenchyma.

LIVER- The liver tissue is acutely congested with areas of autolysis over the surface. Specific inflammation or change or parasitic accumulation is not identified. The degenerative process is occurring secondarily on the surface of the liver.

SLIDE AG- (B10433)
KIDNEY- This tissue is acutely congested with mild autolysis and no evidence of specific other inflammation. Some tubules contain protein. The protein is accumulating secondarily to nonspecific changes in the renal tissue.

LIVER- The liver tissue is acutely congested with very mild autolysis. Minimal inflammation is identified in this tissue. No evidence of parasites is identified in this tissue.

PATHOLOGIST COMMENTS

The mice in this group demonstrate no evidence of specific uniform change. There are large numbers of parasites or parasite eggs in several parts of the liver tissue. The majority of the parasites appear to be Trematodes, although there are some cross sections that are nematodes. The parasites are probably due to a heavy environmental parasitic load in this area. The tissues were all preserved quite well. No evidence of specific inflammation, toxicity or neoplasia was identified in the tissues. Kidneys demonstrated essentially no change except for acute congestion.

07/16/97
(LDM/mdp) Verified by: L. D. McGill, D.V.M., Ph.D., DACVP
                           Veterinary Pathologist
                           electronic signature

For Histopathology Consultation Call: 1-800-426-2099

ESTON REAC, UNKNOWN

END OF CHART

CHAIN OF CUSTODY RECORD

Project Name: _Cornell Dubilier_

Project Number: _03347-142-001-2274-01_

RFW Contact: _JOHN JOHNSON_   Phone: _(908) 321-4200_

No: **05331**

Sample Identification — Analyses Requested

REAC #	Sample No.	Sampling Location	Matrix	Date Collected	# of Bottles	Container/Preservative	Histopathology				
	B10401	T1-14-2	T	17 June 1997	1	AOI 40mL GLASS / Formalin	✓				
	B10402	T1-14-9									
	B10403	T1-8-3									
	B10404	T1-10-6									
	B10405	T1-3-4									
	B10406	T1-14-10									
	B10407	T1-5-5		↓							
	B10408	T1-13-10		18 June 1997							
	B10409	T1-9-7		17 June 1997							
	B10410	#1 T1-14-6		↓							
	B10411	T2-12-8		18 June 1997							
	B10412	T2-4-5		↓							
	B10413	T2-2-7		17 June 1997							
	B10414	T2-3-6									
	B10415	T2-12-3									
	B10416	T2-12-10									
	B10417	T2-2-1									
	B10418	T3-4-5		↓							
	B10419	T3-4-2		18 June 1997							
	B10420	T3-1-8	↓	17 June 1997	✓	✓					

Matrix:

-	Sediment	PW -	Potable Water	S -	Soil
DS -	Drum Solids	GW -	Groundwater	W -	Water
DL -	Drum Liquids	SW -	Surface Water	O -	Oil
X -	Other	SL -	Sludge	A -	Air

T - Tissue (_Peromyscus leucopus_)

Special Instructions:

FOR SUBCONTRACTING USE ONLY

FROM CHAIN OF CUSTODY #

Items/Reason	Relinquished By	Date	Received By	Date	Time	Items/Reason	Relinquished By	Date	Received By	Date	Time
ALL/ANALYSIS	[signature]	6/20/97									

FORM #4

8/94

REAC, Edison, NJ
(908) 321-4200
EPA Contract 68-C4-0022

Project Name: _Cornell Dubilier_
Project Number: _03347-8142-001-2274-01_
RFW Contact: _JOHN JOHNSON_   Phone: _(908) 321-4200_

No: **05335**

SHEET NO _2_ OF _2_

Sample Identification / Analyses Requested

REAC #	Sample No.	Sampling Location	Matrix	Date Collected	# of Bottles	Container/Preservative	Histopathology				
	B10421	T3-1-11	T	17 June 1997	1	40ml Glass/Formalin	✓				
	B10422	T3-4-20		↓							
	B10423	T3-4-1		18 June 1997							
	B10424	T3-3-15		17 June 1997							
	B10425	T3-1-5		↓							
	B10426	T4-23		18 June 1997							
	B10427	T4-1-1									
	B10428	T4-1-27									
	B10429	T4-5-3									
	B10430	T4.2-24		↓							
	B10431	T4-2.2		18 June 1997							
	B10432	T4-3-5		17 June 1997							
	B10433	T4-2-7	↓	↓	↓	↓	↓				

Matrix:

SD -	Sediment	PW -	Potable Water	S -	Soil
DS -	Drum Solids	GW -	Groundwater	W -	Water
DL -	Drum Liquids	SW -	Surface Water	O -	Oil
X -	Other	SL -	Sludge	A -	Air

Special Instructions:

T- Tissue (Paramysis leucopus)

FOR SUBCONTRACTING USE ONLY

FROM CHAIN OF CUSTODY #

Items/Reason	Relinquished By	Date	Received By	Date	Time	Items/Reason	Relinquished By	Date	Received By	Date	Time
All/Analysis		6/20/97									

FORM #4

8/94